Digitized by the Internet Archive
in 2018 with funding from
University of Toronto

https://archive.org/details/medjournalMay1925uoft

The University of Toronto Medical Journal

VOL. II MAY, 1925 NO. 7

EDITORIAL

CHARLATANISM

THE extraordinary prevalence in modern times of chiropractors, osteopaths, naturopaths, and others of like sort, has led to a search for suitable legislation for dealing with the situation. True, it has always been more or less so, and while we have not always had the "electro-therapeuths" and Christian Scientists with us, an older day has seen others just as bad. One doubts whether the time ever existed when a multiplicity of kidney pills and vegetable compounds was not offered to a long-suffering public. Nevertheless, the problem to-day is acute, and we are seeking a solution in legislation. Such legislation is eminently justified, for the state is as much obligated to protect its citizens from exploiters of health as from despoilers of wealth. Yet one sometimes wonders whether the situation can ever be met adequately until something more fundamental is done. To restrict the activities of the rogue is an evident duty, but to protect the public from a system, the dangers of which they do not appreciate, smacks too much of paternalism to thrive long in a democratic state.

The most remarkable aspect of the problem is not the prevalence of irregular healers, but the extraordinary gullibility of those to whom they appeal. Public information on many topics is good, but the most absurd propositions are propounded for the healing of mankind, and gain wide acceptance among intelligent and educated folk. It would seem that this guillibility in the matter of therapeusis is largely due to the monumental ignorance of the average person about his own body and about the methods of modern science. The average man is less informed about himself than about any other object in the universe, and very few, indeed, know that medical science is accumulated experience, and not multiplied theory. Hence, it appears that in education lies the best hope of saving a foolish people from their folly.

But the education must be specific. It must deal with physiology and general biology. Our generation is sorely in need of a modern Huxley to preach the value of biological science in education. It is certainly an unbalanced system which labels a man as "educated" who isn't sure whether or not he has a spleen, and who thinks his blood vessels are all veins. To teach a man the ancient lore of Greece is commendable; to instruct him in the languages of the earth is good; and to point out to

him the significance of the Renaissance is desirable, but he is never fully oriented in his environment until he knows something of himself. The children in our schools are practically never given any instruction in human physiology. They are never told, for instance, that nerves have definite known functions, and do not cause consumption and chilblains when pinched. Indeed, people frequently finish a college course and never seem to grasp the fundamental idea underlying scientific method. These deficiencies in public education explain why so many educated people fall victims to the charlatan's wiles.

Every school child should be taught something of physiology, something of biological science in general. He should be given at least a hint as to the scientific method of observation and experiment. Such instruction need not displace any other essential of education, and it need not be too extensive. Yet it should give the average person some little information about himself and his environment. Such instruction, insufficient in itself, would form a convenient foundation for the further education of the citizen by proper medical publicity, so that people might know at least as much about themselves as they know about locomotives. With a public thus intelligently informed, legislation against charlatanism would have the popular support it deserves, instead of having to be laboriously forced through parliamentary bodies. And what is even more important, the further progress of medical science would be assured of sympathetic support.

HOW TO PASS EXAMINATIONS

A SYSTEMATIC method of answering examination questions is an invaluable aid to the student in overcoming these annual obstacles to his progress. Indeed, a systematic approach to any question in medicine is an essential, whether one is writing, studying, practising, or teaching. Fortunately many medical problems lend themselves readily to such an appeal.

In treatment, for example, we have Dr. Rudolf's arrangement of (1) Diagnosis, (2) Environment, (3) Diet, (4) Symptomatic, and (5) Specific treatment.

Again, any question relating to the pathology or to disorders of an organ can always be most thoroughly answered by keeping in mind the causes of pathology, classified in some such way as (1) Congenital, (2) Traumatic, (3) Toxic, (4) Inflammatory, (5) New growths and cysts, (6) Vascular, (7) Parasitic, (8) Unknown, (9) Functional.

Symptoms may be classified as (1) related to the organ immediately involved, (2) related to neighbouring organs, (3) toxic, (4) reflex, and such an arrangement not only helps one to remember all the symptoms, but forces one to appreciate their true cause, and so indicates the proper symptomatic treatment.

Prognosis should be thought of in two parts, (1) immediate, (2) ultimate.

Diagnosis may be systematized by considering in order, (1) History, (2) Physical examination, (3) Special examinations, (4) Laboratory

Tests and X-ray, (5) Differential Diagnosis. Each one of these may also be systematically approached, especially the first two, whose routine is familiar to us all. With regard to special examinations, however, a routine is less usually practised, but is equally valuable. A suggested routine for the examination of a patient suspected of tuberculosis is outlined elsewhere in this issue. The routine of a thorough neurological, cardiological, genito-urinary and other examinations is equally important to know, and perhaps not sufficiently stressed in our teaching.

The same principles may be applied to all sorts of local examinations, as for example a recent fracture, or a breast suspected of cancer. In all cases the points to look for should be known and brought out in logical order by an established system of examination and questioning. The order in which the various points are considered may be important, but is not so important as that some systematic attack of the problem should be attempted, and that this attack should always be as much the same as possible. Herein lies the foundation of the habits of accuracy and thoroughness.

If lectures and clinics were always given from such a standpoint and with such method, the task of the student would be greatly lightened.

THE FUTURE OF THE JOURNAL

THE completion of the second year of life for THE UNIVERSITY OF TORONTO MEDICAL JOURNAL is a fit occasion for taking stock. At first distinctly an experiment, perhaps not exciting enthusiasm in every mind, THE JOURNAL has, we believe, made many new friends, and increased the confidence of many old ones during these two years. But the record of the past must stand alone, and no remarks which might be made here are capable of altering the verdict as to the merits and demerits of that record. To the future, however, it seems more profitable to turn our eyes, in the hope that a brief but rich experience may aid us in seeing what lies ahead.

We believe that THE JOURNAL will become more and more a medium for the publishing of student articles. In all probability, however, staff articles will continue to occupy an important place among its pages. From the very outset, certain friends advised that THE JOURNAL publish student articles exclusively. This seemed inadvisable, and undoubtedly the generous co-operation of numerous members of the staff has largely determined the success of THE JOURNAL. But this year 43% of the articles were by students, as compared to 20% last year. Thus it is established that a large proportion of the articles can be drawn, and drawn with profit, from students. And further, upon the contributions of undergraduates does the feasibility of the magazine depend.

THE JOURNAL will be a larger magazine in the future. Toward the end of the present academic year, the number of articles in each issue was materially increased, and, as a result, the sales were much better. To such an extent is this true, that within limits it is cheaper to publish a large Journal than a small one. And a more extensive table of contents will surely come nearer to satisfying every reader with something in which he is especially interested.

The circulation of THE JOURNAL will increase. Already many copies leave Toronto, and with a concerted effort to obtain a strong foothold in McGill, Queen's and Western, as well as among recent graduates and other practitioners, the number can be increased. The circulation among recent graduates is already considerable, since THE JOURNAL offers an excellent contact with the student's first source of medical information, no matter where he may live.

Besides these effects which the future will in all likelihood have upon THE JOURNAL, the publication will itself produce some definite results on medical student life at Varsity. The quality of student work will be raised by the encouragement offered to the production of papers on medical topics. The average student's knowledge of medical biography will be increased by a reasonable emphasis on that subject. The student, also, who begins by reading his undergraduate publication will be much more likely to continue reading periodicals after graduation. And finally, the experience gained by students in publishing the magazine can not but tend to make them in every way better educated men.

On the whole, one cannot but be hopeful. Certain difficulties will, of course, be encountered. The difficulty of appealing to the first two or three years is great, since a certain portion of THE JOURNAL is of necessity somewhat uninteresting to them. That problem must be solved by securing a large number of contributions from the men in these years. But THE JOURNAL, we dare to believe, is here to stay, and those of us who relinquish connection with her do so with profound regret, but also with supreme confidence in her future.

INTERNESHIPS FOR WOMEN

The changed attitude toward women in professions, and particularly the increasing willingness of the medical profession to admit women to its ranks, is reflected in the fact that 128 out of 524 hospitals on the approved list now state that they admit women internes. In 1923, 135 out of 510 hospitals on the approved list favoured the admission of women internes. The 128 hospitals now admitting women use a total of 1,385 internes, whereas only about 214 women graduate in medicine each year. Therefore, so far as the number of places open to women graduates is concerned, all should be able to find interneships, and a wide range of choice is provided for them.

—J.A.M.A. 84; 13, 966. (March 28, 1925).

THE ELECTROCARDIOGRAM

J. HEPBURN, M.B.

Demonstrator in Medicine, University of Toronto

Deprived of illustrations, a paper on electrocardiography loses much of its value, and the difficulties of both reader and writer are greatly increased by their absence. In this brief paper only the commoner electrocardiographic abnormalities will be dealt with, and where possible an attempt will be made to indicate their prognostic value.

The inception and much of the development of electrocardiography is the work of the Dutch scientist, Einthoven. Electric currents are developed in the heart, prior to and during the contraction of that organ, and when these currents are led to a string galvanometer, a photographic record of the deflections of the string can be obtained. This photograph is the electrocardiogram, which is composed of three parts, leads I, II, and III, in which the electrodes are connected to the right arm and left arm; right arm and left leg; and left arm and left leg, respectively. The normal electrocardiogram consists of a series of deflections or waves which have been arbitrarily named P, Q, R, S, and T. The P wave is associated with auricular activity and the others are the result of ventricular activity. The reader is referred to the text-books of Lewis, Wiggers, and others for further details about these waves. The timing of events is measured by means of vertical lines on the electrocardiogram. The distance between adjacent lines represents 0.04 second. The distance between the horizontal lines, forming small squares with the vertical lines, represents 1 millivolt. Waves are spoken of as positive when above the base line, and negative when below the line.

(1) The P wave usually has a blunt positive summit, less than 2 millivolts in height. It is often split or excessively high in cases of mitral stenosis. It tends to become negative when the impulse arises low down in the auricle instead of at the sino-auricular node.

(2) The R wave is tall, narrow, and positive—8-10 millivolts in lead I; 14-18 in lead II; 8-10 in lead III. It is preceded and followed by the shallow negative waves Q and S respectively. The width of the base of the R wave is normally less than 0.08 second. Widening beyond 0.10 second indicates block in the passage of the wave of excitation over one of the main branches of the auriculo-ventricular bundle, and will be dealt with subsequently under branch-bundle block. Splitting of the R wave usually accompanies widening but may be present without widening.

(3) The T wave has a blunt summit, about 2 millivolts in height. It tends to become negative in all leads under heavy doses of digitalis, and in evaluating negative T waves, the digitalis factor must be ruled out. A negative T wave in lead III alone is of common occurrence and is of no prognostic value. In fact a positive T wave in lead III can be made negative by drinking a glass of iced water. The sudden appearance of negative T waves in all leads commonly occurs in cases of embolism or

thrombosis of the left coronary artery and these waves gradually become positive as the collateral circulation becomes established, if the patient lives. According to Wiggers, the presence of negative T waves (ruling out digitalis effects), in leads other than lead III alone, carries a mortality of about 60% in 8-12 months.

(4) Sinus arrhythmia is a rhythmic quickening and slowing of the auriculo-ventricular complex, usually speeding up with inspiration and slowing with expiration. It is normally present in children and persists in some adults and is entirely physiological. The arrhythmia tends to disappear as the heart rate increases, *e.g.,* with exercise.

(5) In left ventricular preponderance the main QRS deflection is positive in lead I and negative in lead III. This is commonly seen in cases of arterial hypertension and aortic valve lesions, in both of which the burden of the increased work falls on the left ventricle.

(6) In right ventricular preponderance the main QRS deflection is negative in lead I and positive in lead III. Cases of mitral or pulmonary stenosis in which the excess of work is done by the right ventricle usually show right ventricular preponderance.

(7) Auricular extrasystoles are premature beats arising from some part of the auricle other than the sino-auricular node, and are recognized by the fact that the ventricular waves are similar to the other ventricular waves, but occur prematurely and are followed by a compensatory pause. The P wave, if not buried in the previous T wave, is often abnormal in shape.

(8) Ventricular extrasystoles are premature and are usually followed by a compensatory pause, but the QRST waves are always of different shape from the normal waves for the individual. Extrasystoles, *per se,* do not mean myocardial disease and they tend to disappear when the heart rate is increased.

(9) Auricular fibrillation is characterized by the absence of P waves and the absolute irregularity of the ventricular rhythm. The ventricular rate may be slow, below 40, or fast, up to 200 per minute. Auricular fibrillation always means damage to the myocardium, toxic or otherwise. The presence of extrasystoles in cases of auricular fibrillation is said by Norris to make the prognosis as serious as that in pulsus alternans, *i.e.,* 90% mortality in 18 months.

(10) In auricular flutter the ventricular rhythm may be regular or irregular, while the auricular (P) waves occur regularly at a rate, (usually over 250 per minute) much faster than the ventricular rate. Each ventricular complex follows a P wave at a definite time interval.

(11) Paroxysmal tachycardia is usually of auricular origin, but may be ventricular. In both cases the impulse arises ectopically, *i.e.,* away from the sino-auricular node, and the rate is usually between 150 and 250 per minute. In auricular tachycardia all the waves can be identified, but the P waves (at least) are different in shape from the normal P waves for that individual. In ventricular tachycardia the ventricular complex is of abnormal shape and if P waves are present they cannot be identified as they are buried in the ventricular waves.

(12) Prolonged auriculo-ventricular conduction time. The maximum normal conduction time of PR interval, measured from the beginning of the P to the beginning of the R wave is 0.20 second, with an average of 0.16 second. An increase in the PR interval beyond 0.20 second indicates a delay in conduction over the auriculo-ventricular bundle and, ruling out digitalis therapy, always means disease of the heart muscle, toxic or otherwise.

(13) Incomplete heart block is a further step in faulty conduction. In this condition the PR interval is greater than 0.20 second and there will be seen one or more P waves with no ventricular waves following, *i.e.,* there will be dropped beats.

(14) Complete heart block is recognized by the complete dissociation between the P waves, which occur regularly at their normal rate, and the ventricular waves, which occur regularly, but at a much slower rate—about 30 per minute. Cases with a much quicker ventricular rate are not uncommon and are usually not suspected clinically. There is always myocardial damage in cases of complete and incomplete block.

(15) Intraventricular or branch-bundle block. The auriculo-ventricular bundle divides into two main branches, one of which passes down on each side of the interventricular septum towards the apices of the right and left ventricles respectively. When the wave of excitation fails to pass along one of these branches, the resulting condition is called branch-bundle block, which gives a very characteristic electrocardiogram. The branch to the left ventricle divides immediately after its origin, while the right branch does not divide until its termination. Consequently an extensive lesion is required to block the left branch while a very small lesion will block the right branch. As a natural result of this, cases of right are much commoner than cases of left branch-bundle block.

Right branch-bundle block is recognized by (1) a widened R wave—the base being more than 0.10 second (the normal width being less than 0.08 second; (2) the T wave in each lead being in the opposite direction to the main QRS deflection; (3) the presence of left ventricular preponderance. Occasionally there is no preponderance.

The picture in left branch-bundle block is similar to that in block of the right branch in every detail, except that in this case there is right ventricular preponderance. The prognosis of branch-bundle block is very grave and the generally accepted statement is that 90% of these cases are dead within 18 months.

(16) Low voltage, *i.e.,* very low waves in all leads, is said to indicate definite and usually serious myocardial damage.

(17) Pulsus alternans, *per se,* shows no electrocardiographic abnormality as a rule, but occasionally some part of the ventricular complex (QRS or T waves) may show alternating changes in height.

The value of the electrocardiogram from a diagnostic, therapeutic, and prognostic standpoint, lies chiefly in the detection of the following abnormalities which cannot be recognized clinically:

(1) Branch-bundle block with its 90% mortality in 18 months.

(2) Negative T waves which have a bad prognosis when not due to digitalis.

(3) The differentiation between auricular fibrillation, auricular flutter with an irregular ventricle, and frequent irregularly occurring extrasystoles. This is important as the treatment and prognosis are different in the three cases.

(4) Prolonged conduction times gives no clinical indication of its presence. Digitalis should be administered with care in this condition.

(5) Complete heart block, when the ventricular rate is fairly rapid, is rarely suspected clinically.

(6) Extrasystoles in a case of auricular fibrillation cannot be detected clinically, and are believed to have a very serious prognosis.

Heart consultants hesitate to give a good report on a patient beyond middle life even in the absence of definite signs or symptoms of heart disease, without seeing an electrocardiogram of the patient.

The mortality statistics of the Department of Medicine confirm, with some minor differences, the seriousness of the abnormalities mentioned above.

FROM THE DOCTOR'S DIARY

In a book of extracts from English diaries edited by Arthur Ponsonby, are some doctors' diaries that make very interesting reading. Dr. John Dee, born 1527, who apparently was more magician than doctor, and held conversation with spirits, makes the following note after an attack of renal colic.

"A great fit of stone in my left kidney; all day I could do but three or four drops of water but I drunk a draught of white wyne and salet oyle and after that crabs' eyes in powder with the bone in the carp's head and about four of the clok I did eat toasted cake buttered and with sugar and nutmeg on it and drunk two great draughts of ale with it and I voided within an hour much water and a stone as big as an Alexander seed. God be thanked."

Dr. John Rutty in the middle of the 18th Century writes an unconsciously humorous and most human diary, wherein he records his struggles with his besetting sins of quick temper and overeating. His entries are very brief and to the point.

"This day an acquaintance broke deeply in my debt; the deception great and the loss considerable: Lord Thou hast smitten: sanctify the event."

"Take care, take care of the fumes of cyder and whiskey, tremble at the mixture."

"I feasted pretty moderately; but with this notable difference in solitary and social eating, that in the last I eat more like a swine."

"A little incubus last night on too much spinach."

"Snappish. Lord shall this sin which so easily besets be never, never overcome?"

"A little crabbed with two incurables."

"The medical profession exhibits strongly the vanity and wickedness of the world where the more work the less pay."

"Eleven visits and no fee; blessed be the Lord."

THE DIAGNOSIS OF PULMONARY TUBERCULOSIS

T. G. Heaton, '25

(Abbreviated from a paper read before The Seventeen Club, October, 1924)

In this paper the routine examination of a patient suspected of pulmonary tuberculosis is described, and the diagnosis of clinical activity, the differential diagnosis, and the prognosis are briefly discussed.

The most plausible stand to take on the development of pulmonary tuberculosis in a patient would seem to be as follows:

1. The original infection is received in childhood by inhalation, ingestion, or inoculation.
2. The source of infection is the open case of tuberculosis.
3. Bovine infection rarely or never causes phthisis, but confers immunity.
4. The childhood infection produces a permanent immunity to exogenous re-infection—except probably to re-infection with massive doses, and this rarely occurs.
5. Phthisis is an exacerbation in predisposed individuals of the latent childhood infection.

The problem of phthisiogenesis becomes, therefore, one of predisposition. The following are generally accepted as predisposing causes of phthisis: poverty, certain anatomical peculiarities, (*e.g.,* stenosis of upper thoracic aperture) frequnet colds or sore throats, measles, scarlet fever, whooping cough, diphtheria, occupation, (*e.g.,* involving inhalation of dust), injury to chest, malnutrition, overwork, worry, and focal infections resulting in poor health. These factors must be remembered in making a diagnosis and taking a history.

It will now be plain that what is wanted is not "early" diagnosis of pulmonary tuberculosis, for the lesion has probably existed since childhood, though it may not become active till old age, and to demonstrate an inactive lesion in adolescence is not particularly desirable. But what is wanted is a *prompt* diagnosis of clinical activity. For a similar reason, it is incorrect in most cases to speak of an "early" lesion simply because the physical signs are here slight. This "early" lesion so called, may be a fibroid lesion of long standing—and small cavities may even be present. Therefore in diagnosis our aim should be the *prompt* recognition of *minimal* tuberculosis, recognizing that even the minimal lesion may be one of long standing. On the other hand, it is not safe to say that when a far advanced lesion is discovered, a long period of clinical activity is behind it, for in some cases there is no stage of early activity to be recognized and the disease has involved a large area of lung all at once, as in cases of spread by aspiration.

The ideal before us then is stated as the prompt diagnosis of minimal tuberculosis. How are we to suspect the presence of phthisis? What symptoms will bring a tuberculous patient to us?

We must suspect pulmonary tuberculosis when there is cough, loss of weight, hoarseness, digestive disturbance, neurasthenia, anaemia, fever and rapid pulse, sweating, chills, rheumatic pains unrelieved by salicylates, pro-

tracted convalescence, ischio-rectal abscess, and especially hemoptysis and pleurisy.

Lawrason Brown of Saranac states that "In any patient with constitutional symptoms, no matter of what he complains, the possibility of tuberculosis must always be kept in mind".

After our suspicions are aroused our problem really commences. Has the patient got tuberculosis? If so, to what extent? What are his chances of recovery? These are the questions that an accurate diagnosis must answer.

The History

The first and most important step in diagnosis is the history taking. Louis Hamman goes so far as to state "I am convinced that early diagnosis would profit if physicians could be rid of the bugbear of physical examination, which exacts that every diagnosis be supported by gross changes in auscultation and percussion".

A family history of tuberculosis or history of exposure in childhood should be inquired into—also whether the patient was breast or bottle fed. Nationality should be ascertained—the Celtic race, for example, has a low resistance to tubercle. Occupation is important as a predisposing cause, and in the personal history and history of past illnesses, other predisposing causes, such as hygiene of environment, overwork, worry, and diseases or focal infections possibly predisposing to phthisis should be ascertained. The history should be carefully kept in chronological order in its various sections, and especially in the section on present illness, a complete history of each symptom should be secured, and careful inquiry made as to the occurrence of symptoms known to the doctor to be suggestive of phthisis, such as ischio-rectal abscess, pleurisy, hemoptysis, night sweats, cutaneous flushing, easy fatigue, and increased nervousness. The weight is important and the history should include the patient's best weight with date, his usual weight and his lowest weight in his present illness, with date.

The history should close with an analysis of the patient's present condition by systems. In women a careful menstrual history should be obtained. Menstrual disorders are very frequent in phthisis. A well taken history is often sufficient for diagnosis alone, and is always of great value in prognosis.

Physical Examination

This should be conducted as a complete general examination, but the proper examination of the chest involves details not practised by many of the profession, so that it will not be out of place to describe the physical examination here. Most weight is placed upon auscultation, with inspection next. Refinements of percussion to the extent that some claim possible are not likely to be reached by the average man.

The first step is superficial examination, observing colour of face, condition of scalp, external ear, eyes, cervical glands, thyroid, mouth and teeth, then of the chest. The patient should be seated in a good light, stripped to the waist, and preferably on a revolving stool, as a piano

stool. The movements of the chest are observed both from the front, and with the examiner standing behind and looking down on the front of the chest while the patient breathes deeply. Lagging and limitation of movement are to be observed. Note also, drooping shoulder, curvature of spine, prominence of clavicles, form of chest, rib obliquity, and muscular atrophy. A spinal curvature may account for slight differences in apical percussion. Then comes percussion. Kronig's bands, the excursion of the lungs in the scapular line, the heart borders, and the point of maximal impulse are first determined and measured. It is well to auscultate the heart at this stage lest it be forgotten later. Then the lungs are systematically sounded all over, front and back, being careful to compare symmetrically opposite points. It is useful first to find a zone of normal resonance, one on each side, usually under the axilla, for purposes of comparison. Percussion over the front of the chest should be gentle first, then deep, for gentle percussion is necessary to detect superficial lesions, but heavier percussion will be necessary for the detection of deeper changes. At the back, heavier percussion is necessary everywhere. A useful trick of percussion is expiratory percussion. The effect of full expiration is to increase resonance over healthy lung and to diminish it over diseased lung, so that a difference hard to ascertain is reinforced by percussion in full expiration. Particular care should be taken, both in percussion and auscultation, in examining the "points of election" of tuberculosis, namely, beneath the junction of the middle and outer thirds of the clavicle in front, *i.e.*, the apices, and the apices of the upper and lower lobes at the back, *i.e.*, opposite the 1st and 3rd Dorsal Spines, at the edge of the vertebral border of the scapulae when the shoulders are dropped forward. Riviere says that to find impairment to percussion at more than one point of election is almost diagnostic. The points to look for in percussion are, of course, dullness or hyperresonance, limitation of excursion, displacement of mediastinum (as in pneumothorax) cardiac enlargement, and asymmetry or blurring of Kronig's bands. It is well to remember that pleural thickening may be a cause of dullness.

Palpation is of slight value—vocal fremitus is not usually elicited, and the muscle spasm which some workers report is rarely discovered and is not reliable. Auscultation is the next and main method of examination, but it must be remembered that there is nothing absolutely characteristic about the adventitious sounds in phthisis. For diagnosis the essential thing is that they must be permanent and localized to the same area on repeated examination. They must be tested for, first, with quiet breathing through the mouth, then during forced breathing, and then during the inspiration following a cough at the end of expiration. Lawrason Brown states that "the detection of rales by the auscultation of the inspiration following cough is the most important procedure in the detection of physical signs in pulmonary tuberculosis".

It is best first to find a normal vesicular murmur, usually in the axilla, and then to go all over the chest listening to breath sounds only, then go over the chest again, still with quiet breathing, to listen for rales, then with forced breathing, then to listen to the inspiration following cough, and finally to listen for whispered bronchophony. Experience may shorten

this procedure. Special attention should be paid to the points of election, the supraclavicular spaces, and areas high in axilla—placing the bell of the stethescope beneath the edge of the pectoralis major.

The patient is then made to lie down, and the abdomen examined, and the remainder of a routine physical examination carried out in the usual way. After this, the ear, nose, nasopharynx and larynx are examined by mirror and speculum, and if possible the sinuses should be trans-illuminated. This completes the physical examination, except for special examinations in special cases.

It is easy enough to go through this form of examination. It is harder to interpret what we hear. Take changes in the breath sounds for example. Osler held that a feeble breath sound or harsh respiratory murmur was a sign of minimal tuberculosis. But feeble breath sounds mean that the lung does not breathe as freely as usual, *i.e.,* there are adhesions, or it means a thickened pleura—and in either case denotes an old lesion. An increase in the bronchial element of the breathing, it will be remembered, means increased conductivity of the lung from either fibrosis, or collapse, or consolidation—*i.e.,* an old lesion. It is generally agreed now that the earliest sign of activity is rales—fine rales, that persist or are increased after cough. Rales without altered breath sounds therefore, must mean a recent lesion. Rales may, of course, be present in company with the signs of an old lesion. The rales of active, pneumonic, parenchymatous disease, are fine, moist, crepitant rales, heard in showers, and uniform in size on inspiration only. Chronic lesions have a larger rale, and dry rales probably indicate an inactive lesion, or scar.

The so called atelectatic rales heard on first deep inspiration, at the apex usually, and disappearing after cough, are without serious significance. No rale which disappears after cough is tuberculous.

Sounds resembling rales are: (a) Marginal sounds heard at the bases in health, caused by separation of pleural surfaces on the excursion of the lung. (b) Sounds originating in costo-sternal, or sterno-clavicular articulations—especially in athletes. (c) True rales may be transmitted to the opposite side of the chest and heard more faintly there. This must always be watched for in cases where rales are heard on both sides. (d) Skin sounds and hair sounds are annoying and confusing, and may be largely eliminated by using a bell stethescope and by vaselining the hair. (e) Muscle sounds.

The signs of cavity, it will be remembered, are amphoric breathing, musical or consonating rales, and pectoriloquy; but cavities may be present without these signs.

It is a popular superstition that the coin test is very commonly, or even invariably heard in cases of pneumothorax, but this is the exception rather than the rule. Pneumothorax is much better diagnosed by other signs, especially by feeble or absent breath sounds and displacement of the mediastinum. In some cases the apex beat may be seen to the right of the sternum.

The essential things in making a diagnosis of phthisis from physical examination are:

1. Abnormal findings, whether altered breath sounds change in per-

cussion note or rales, must be persistent and localized on repeated examination.

2. Rales, to be significant of tuberculosis, must persist after cough. The careful examination over the whole chest, of the inspiration following cough is therefore essential.

Either immediately on the completion of the chest examination, or during the examination, the findings should be charted or set down on paper in some way. Conventional signs for noting physical findings are in common use and should be employed. They provide a shorthand, accurate method of writing down one's findings, and give a complete picture at a glance to a succeeding examiner of the condition of the patient's chest at a former examination.

Laboratory Tests

From history and physical examination the diagnosis in the majority of cases can be made with reasonable certainty, but many cases, especially minimal cases, will be met with, which cannot be diagnosed by these means alone. Indeed it is such a serious thing to diagnose tuberculosis, that all established means of diagnosis should be tried in any case. By "established" I mean to exclude tuberculin tests and serum reactions. Their place in diagnosis will be discussed. In addition to history and physical examination, the other aids to diagnosis fall readily into two groups of greater and lesser importance. The first and most important group, which should be used in every diagnosis of phthsis, consists of sputum examination, radiography, and observation of the temperature and pulse. The second and less important group consists of the tuberculin tests, serum reactions, and a few other tests. We shall take the first group first.

The Sputum.

Ten or even twenty sputum examinations are not too many in a case suspected of phthisis, indeed no number of negative examinations can be said to exclude phthisis. The sputum may be examined by direct smear, and acid fast staining technique, but cases negative by this method should always be examined by one of the concentration techniques. That recently introduced by Andrus of Byron Sanatorium, London, Ont., is one of the best. It certainly gives excellent results and has peculiar advantages.

If there is no sputum, mucus mopped from the laryngeal region may be found to contain the bacilli.

The examination of the sputum is complicated by the existence of non-acid fast, but virulent, tubercle bacilli, called "Much staining bacilli", and of acid fast avirulent bacilli of various sorts.

In cases of doubtful diagnosis in whom acid fast organisms are found in the sputum, the virulence of these organisms should always be ascertained, by staining for smegma, or usually, by guinea pig inoculation with the sputum.

Elastic fibres in the sputum denote only advanced destruction of lung tissue.

The presence or absence of secondary organisms in the smear should be noted.

Radiography

It is certain that no chest examination is complete without stereoscopic X-ray plates of the chest. The single plate is of comparatively slight value, and is much harder to interpret.

The interpretation of X-ray plates is a matter for experience rather than explanation. The best short article I know on the subject is that by Kennon Dunham in the American Review of Tuberculosis of Nov., 1918, in which the well-known "Dunham's Fans" are described. In general one may say that increased density in the upper third of the lung should be considered tuberculous till shown to be otherwise, increased density in the lower third should be considered non-tuberculous till shown to be so, and increased density in the middle third is interpreted according to the findings in the other two-thirds.

The chest may be examined under the fluorescent screen, for failure of the apex to brighten is an early sign of trouble. An area of tuberculosis sufficient to cast a definite shadow, however, is a well advanced lesion. Dunham's article gives also the technique of screening.

The temperature

The observation for two weeks of the temperature and pulse taken every 3 hours during the day may be a considerable aid.

Other Tests

We now come to the group of tests less important in diagnosis, namely, the tuberculin tests, the complement-fixation test, and the inhibitive reaction. Space prevents a discussion of their technique and interpretation here. Their place in diagnosis is that of an added aid in difficult cases.

Differential Diagnosis

We have now completed our examination of a suspected case of phthisis. This should have included, (1) A careful history. (2) A complete physical examination, including examination of the ear, nose and throat and sinuses. (3) Several sputum examinations. (4) Stereoscopic X-ray plates of the chest. (5) A temperature and pulse record for several days. (6) Any other tests that seem necessary if a diagnosis cannot be reached on the first five.

With the data so obtained before us we must make our diagnosis, and the differential diagnosis of tuberculosis is often difficult.

The conditions most likely to give difficulty in the diagnosis of phthisis are: apical pulmonary collapse, chronic bronchitis, pulmonary oedema in mitral stenosis, bronchiectasis, asthma, syhpilis of the lung, hyperthyroidism, upper respiratory disorders, cancer of lung, anaemia, obscure fever, pulmonary abscess, chronic empyema, unresolved bronchopneumonia, mycosis, and pneumokoniosis.

Of these we will only mention briefly upper respiratory disorders. These may cause local atelectasis and peribronchial thickening. For example, to quote J. S. Pritchard of Battle Creek, "A partial obstruction to

the inspired air, caused by a deflected septum, enlarged tonsils, adenoids or polypi, may lead to abnormal physical findings in the thorax due to a localized atelectasis in one or both apices and in such instances a physical examination of the chest may reveal deficient expansion, feeble vocal fremitus, weakened breath sounds, often grouped, persistent rales, and sometimes definite apical retraction." The importance of a nose, throat and sinus examination will now be appreciated, for the signs given above resemble accurately those of phthisis and may yet be caused by an upper respiratory disorder.

We have now been able to decide that our patient has or has not got phthisis. If he has, we must know to what extent and must be able to give him a prognosis.

One of the biggest problems in the diagnosis of phthisis is the determination of clinical activity, *i.e.*, whether the lesion which we have discovered, possibly in a routine physical examination for some other complaint, is active or not. Lawrason Brown states that symptoms are a better and more reliable guide to activity than physical signs. He regards as signs of activity a temperature always over 99 in a male and 99½ in a female, or a pulse frequently over 90 in a male and 96 in a female, or a combination of lassitude, loss of weight, pleurisy, night sweats, etc. On the other hand, expectoration, cough, tubercle bacilli in the sputum, dyspnoea or hoarseness, in the absence of other signs, he does not consider signs of activity.

Prognosis

The prognosis is the last, but not the least, part of diagnosis. It is, after all, the only part of interest to the patient, and it behooves the doctor to be sure of his ground. Lawrason Brown gives the following prognostic aphorisms.

"An acute onset points to prolonged illness or early death."

"Continuous gain in weight on an ordinary diet is the best indication of favourable progress, but can occur with advancing disease."

"Digestion is the keystone of the prognostic arch."

"Pulse, temperature, and weight form the prognostic triad."

"Extent of disease postpones arrest longer than localized intensity."

"The condition of the opposite lung is important."

"Puberty and menopause have less bearing upon the disease than pregnancy, especially repeated pregnancies, and hence marriage for women increases uncertainties." Other authorities say that no woman with phthisis should marry, or at any rate should not bear children or give suck.

Summary

We have now come to the end of a long paper that has nevertheless dealt too briefly with its subject. I shall sum up by repeating a few points I want to emphasize.

1. Minimal tuberculosis is hard to diagnose.

2. The history is the most important part of diagnosis, then the physical examination. The detection of rales by inspiration after cough is the most important part of physical examination.

3. Sputum examinations must be frequent if necessary, and never neglected.

4. The stereoscopic X-ray is an essential to proper chest examination, it always shows a lesion greater than physical signs would indicate.

5. Abnormal physical signs in the apex should be considered as due to pulmonary tuberculosis till proved not to be, and those at the base should be looked on as non-tuberculous till proved to be tuberculous.

6. "In any patient with constitutional symptoms, no matter of what he complains, the possibility of pulmonary tuberculosis must be kept in mind."

7. Hemoptysis and "idiopathic" pleurisy should be considered tuberculous till proven not to be so.

References

1. Fishberg: Pulmonary Tuberculosis.
2. Rivière: Early Diagnosis of Pulmonary Tuberculosis.
3. Osler: Principles and Practice of Medicine.
4. Parfitt:Prompt Diagnosis of Pulmonary Tuberculosis.
5. Bushnell: Diagnosis of Tuberculosis in the Military Service. A. R. of T., 1917.
6. Parsons, Elliott and Caulfield: The Diagnosis of Pulmonary Tuberculosis.
7. Louis Hamman: The Diagnosis of Pulmonary Tuberculosis. A. R. of T., June, 1917.
8. Pritchard: Changes in Lungs and Bronchi due to upper Respiratory Disorders.
9. Lawrason Brown: Trudeau Studies.
10. Kennon Dunham: A Manual of the Roentgenological Examination of the Chest. A. R. of T., Nov., 1918.

SPRAINS*

HAROLD W. WOOKEY, M.B., F.R.C.S., (ENG.)

Demonstrator in Clinical Surgery, University of Toronto

DEFINITION

A sprain is an injury to the soft tissues of the locomotor system produced by stress, the result of sudden violence directly or indirectly applied.

Such injuries are commonest in the neighbourhood of joints, because of the great range of movement and the fixation of capsule and ligaments close to the articular lines.

AETIOLOGY

Sprains are the result of either excessive, unguarded or abnormal movements.

Movements of joints are controlled by muscular action and guarded by muscular tone. As a result, normally a minimum of strain is thrown on the capsule and ligamentous structures which constitute a second line of defense. When the strain is excessive, the muscular protection is overcome. The muscle usually relaxes, or may tear in some part of its structure, and the strain is then borne by the non-elastic ligamentous tissue. If the force is sufficient, ligaments, capsule, and synovial membrane are torn, the degree varying from rupture of a few fibres of a ligament to a gross laceration of the capsule and synovial membrane. The ligaments and capsule of a joint do not stretch on sudden stress, but may after the strain is prolonged on the tissues altered by inflammatory reaction. If the movement is unguarded the strain is at once thrown on the fixed tissues. Abnormal movements, such as torsion of the knee, are always quite unprotected by muscular action, and a comparatively slight force will cause a sprain.

PATHOLOGY

When a strain occurs, blood is always extravasated into the tissues about the joint or into the synovial cavity itself. This is usually followed by a simple inflammatory reaction, which in the majority of cases results in complete repair of tissue and restoration of function. When improperly treated, recurring attacks of pain, persistent weakness or limitation of movement result from non-union of the torn fibres and adhesions in or about the joint. Tuberculous disease or some form of arthritis may follow such injury.

The Diagnosis, after fracture has been eliminated, depends on tenderness, pain, limitation, and occasionally abnormal movement with signs of extravasation of blood.

*Published by courtesy of the University of Toronto Medical Bulletin.

Treatment

As a sprain is a solution of continuity of soft tissue, just as a fracture is of bone, the treatment is essentially the same.

The joint should be kept at rest by strapping or splinting in a position which will relax the injured structures and permit union by the torn fibres. The rest and position should be maintained until Nature has had time to repair the lesion, from a week in minor cases, to six weeks in such severe lesions as sprain of the internal lateral ligament of the knee, with its probable complication of involvement of the medial memniscus.

If seen early, pressure as by bandaging will limit effusion and swelling. Cold applications will relieve pain. After the required period of fixation has elapsed, active movements and massage for the restoration of function must be undertaken. Alternate hot and cold baths are useful. Passive manipulation may be employed, but not beyond the range of painless movement. Measures to protect the healing tissues must be maintained until recovery is complete. Where practicable the nutrition of the muscles should be maintained by electrical stimulation or massage in the early stages when the joint is fixed.

Sprained Ankle

The lesion commonly involves one of the fasciculi of the external lateral ligament. To secure apposition of the torn structures, the foot must be everted and splinted in this position by adhesive strapping or Plaster of Paris.

Absolute rest is essential for a week or ten days, after which the patient may be allowed to get about again. The eversion must still be maintained by the use of a boot, the sole and heel of which is slightly thickened on the outer side.

Conversely, if the internal lateral ligament is damaged, the foot must be inverted and splinted in this position. Later, the newly united tissue must be protected by thickening the heel and sole of the boot on the inner side.

Sprains of Knee

Sprains of the knee are commonly the result of twists, and most frequently involve the internal lateral ligament.

This structure consists of fibres of varying lengths. The innermost are comparatively short, being fixed to the bones close to the articular lines, and also to the outer border of the internal semilunar cartilage. The outer fibres are much larger and are attached to the bones at some distance from the articular surfaces.

The inner fibres gives way first and the semilunar cartilage is frequently involved. Most commonly the anterior attachment is torn, but the injury sometimes includes both anterior and posterior attachments. The cartilage may be torn across the middle, or split longitudinally.

When the internal lateral ligament is torn, the strain is next thrown on the crucial ligaments, which may rupture, or the tibial spine may be broken off.

Such severe lesions are accompanied by extensive haemorrhage into the joint. The slighter injuries are frequently followed by a simple serious effusion into the joint.

In the slight cases, a short period of rest and splinting will effect a cure, but in the more severe injuries at least six weeks of absolute rest is essential.

Effusion should be treated by pressure, and in some cases by aspiration. Haemorrhage increases the possibility of adhesions. A plaster splint extending from the upper part of the thigh to the ankle gives excellent fixation. When the splint is removed, general measures for restoring function of the joint must be carried out. The injured ligament must be protected from strain by thickening the inner aspect of the sole and heel of the boot.

Sprain of Wrist

For sprain of the wrist, rest in the dorsi-flexed position of the hand and subsequent massage are alone required. Radiography has shown, however, that such conditions are often associated with fracture or dislocation of a carpal bone. Occasionally a painful weakness follows from non-union of fragments, or an unreduced dislocation, and the removal of the fragment will be necessary.

Sprains of the Spine

Sprains of the spine are common accidents, and most frequently affect the cervical and lumbar regions.

In the simpler cases, damage is confined to the external muscles and ligaments, and a short period of rest in bed, followed by massage, will suffice to cure.

In severe injuries, the ligamenta subflava are torn, the spinal canal is opened, and pressure from haemorrhage may lead to a temporary or permanent paraplegia. A prolonged period of rest in bed is essential. Subsequent support by spinal brace or plaster may be needed in severe types.

RECENT KNOWLEDGE OF SPLEEN

Reviewed from Dr. Joseph Barcroft, The Lancet, Feb. 14, 1925

Prof. Barcroft and his associates have recently contributed some monumental researches upon the physiology of that previously poorly understood organ, the spleen. Their attention was first attracted by observations made a few years ago, while on a trip to the Andes for the purpose of studying the mechanism of acclimatization to high altitudes. For this purpose it was necessary to know the normal, sea-level blood-volume of each of the members of the party. Determinations made regularly throughout the voyage revealed the astonishing fact that the blood volume progressively increased as the equator was approached, to fall again as it was left behind. A rise of total haemoglobein was also noted, so the increase was cellular as well as fluid. The physiological significance was clear enough; "large cutaneous vascular areas are

opened up in response to the heat. The blood to fill them must come from elsewhere, and as 'elsewhere' may have need of it, it is desirable to have a volume of blood commensurate with the size of the vascular bed." Similar observations were made on persons living for short periods in overheated rooms on the return to England. The increase was too rapid to be explained by new blood formation and there was nothing in the blood picture to indicate such a process.

It was found that in resting animals subjected to low partial pressures of CO the erythrocytes in the spleen contained no COHb even after several hours, while those in the general circulation became, in a few moments, equiliberated with the atmosphere. If the partial pressure of CO were raised sufficiently, COHb appeared in the spleen, but only after 20 minutes, and on removing the animal to the fresh air, while the blood in the circulation rapidly lost its CO, that in the red blood cells in the spleen remained for upwards of an hour.

In struggling animals, on the contrary, the CO penetrated and left the splenic erythrocytes synchronously with those of the blood stream.

Efferent nervous impulses, originated for example by anoxaemia, were clearly shown to cause splenic contractions with the liberation of a material largely composed of corpuscles.

X-ray determinations showed that the living spleen in the cat may be four times the size of the post-mortem room spleen and about 1% of that of the animal.

As a result of repeated haemorrhages the spleen was made to contract to one-quarter its normal size and to a smaller weight than even the dead spleen, moderate exercises resulted in the same changes, but to a lesser degree. Sufficient corpuscles are extruded under these circumstances to increase the blood volume by one-quarter to one-third.

Experiments were performed to determine whether the possession of a spleen really made any difference to the organism. Guinea-pigs of three categories were exposed to coal gas. (a) Normals, (b) those in which the abdomen had some omentum or pancreas removed, and (c) those which had been splenectomised. The mean percentage of COHb in the blood at the time of death was the same in all three groups (83-5%). The longevity of the operated control animals was reduced by 7%, that of the splenectomised by 26%. When similar animals were exposed to hydrocyanic acid gas, which does not poison by making a demand on the haemoglobin in the circulation, there was no difference in the longevity of the groups.

Prof. Barcroft's conclusions are that this work does not condemn current knowledge as regards the functions of the spleen, but renders it more intelligible. "The blood volume should be regarded not as aliquot part of the body weight, but as a physiological variable which is adjusted to the work required of it, and to the size of the 'bed' which it occupies." The spleen should be regarded "not a 'ductless gland', but a definite part of the vascular system". "It has a function entirely in conformity with its muscular structure, being, in fact, a reservoir of corpuscles at once fitted by its reticulum to detain them, or by its musculature to expel them when required to do so." As a result he feels that "we are enabled to arrive at an intelligible estimate of when and why the body is likely to 'vent its spleen'."

RECENT ADVANCES IN THE PHYSIOLOGY OF URINARY SECRETION

J. Markowitz, M.B.

Demonstrator in Physiology, University of Toronto

In discussing the mechanism for the formation of urine, it is customary to enunciate three theories of renal secretion:

(a) *Bowman and Heidenhams theory* states that the glomerular capsule permits the transudation of water and readily diffusible salts, and the tubules add the other organic and inorganic constituents found in the urine. The kidney thus manufactures urine just as the pancreas makes pancreatic juice. Water on its way from blood-vessels to gland lumina, picks up the products of secretion by certain specialized cells.

It is practically certain that this view in its entirety is incorrect. The theory, when postulated, had little more than anatomical evidence to support it. The onus of disproving it rested, therefore, with those who assailed it. By imagining various functions to reside in different parts of the glomerulus and tubule, the adherents of this theory had enough permutations and combinations to explain all the known abnormalities in the composition of the urine. It is probable that there is some truth in this hypothesis, as a certain amount of evidence exists that the tubules may secrete material into the lumen. The theory has many serious objections. Even the grosser details are nebulous, ascribing as they do, mathematical intelligences and powers of minute discrimination to individual cells. It is correctly designated, therefore, as the *vital* theory.

(b) *Ludwig's theory* for a considerable time rivalled that of Bowman and Heidenham. It held that the capsule of Bowman was merely a filter which allowed plasma, minus its protein, to filter through. In the tubules this dilute filtrate was concentrated, the bulk of the fluid returning to the blood by a process of diffusion. No known physical force, however, can form from blood plasma a fluid of considerably greater osmotic pressure, like urine. This theory of Ludwig's has served, in consequence, as a scaffold for what Cushny calls, the *modern* theory.

(c) *The modern theory.* Realizing that the physical factors alone will not account for reabsorption in Ludwig's theory, the modern theory is compelled to resort to a vitalistic concept. According to this theory the formation of urine is a dual affair, each moiety of which differs in mechanism and in location. The glomerulus functions passively as a filter, the filtrate consisting of deproteinized blood plasma. The filtrate is a sort of Locke's solution, and in its passage down the tubule becomes concentrated by a *selective, vital activity* of the tubule epithelium. Those constituents which are not reabsorbed, irrespective of their concentration in the glomerular filtrate, are termed *non-threshold bodies.* On the contrary, those which are reabsorbed only when they exist below a certain concentration in the filtrate, are termed *threshold* bodies. Urea is an example of the former, glucose of the latter. The teleologist would enunciate the theory differently: The kidney tubule rejects substances

which are of no use to the organism, reabsorbing those which the blood requires to keep its composition constant. Although the meaning of these two propositions is identical, the less one ascribes cognate sensibilities to renal units, the clearer will be our conception of the *modern* theory. For example, egg-albumin when parenterally introduced into the organism is promptly excreted. The glomerular capsule is evidently permeable to egg-albumin. This is preferable to saying that the glomerular capsule senses the difference in composition between blood albumin, and egg albumin, and rejects the latter. In seeking to explain natural phenomena, Socrates admonished that we omit the gods. We must be careful not to relegate these gods to individual cells, who may, as it were, act for them by proxy.

According to this theory, then, *threshold* bodies are reabsorbed in definite proportions, which are determined by their normal concentration in the blood. When the concentration of sodium chloride in the blood is low, as in lobar pneumonia, acute rheumatic fever, almost no chloride appears in the urine; when the concentration of sugar in the blood is high, sugar appears in the urine.

The modern theory has recently received its best confirmation at the hands of Richards, and his colleagues, at the University of Pennsylvania. Their methods were prompted by the investigations of Krogh on the capillary circulation. Krogh focused a powerful beam of light on the resting muscles of frogs and guinea-pigs, *in situ*. The illumination so obtained was sufficiently powerful to enable him to observe the behaviour of the capillaries with a binocular microscope. It occured to Richards and his collaborators that the use of the same method might enable them to see the glomeruli of the kidney in operation, providing that they could find a kidney sufficiently transparent to permit the penetration of light rays, The frog's kidney fulfilled this demand, and the results obtained by this method have been very important.

They found that the blood flow in a given glomerulus was not continuous, but intermittent, so that a glomerulus would pulsate for fifteen seconds or so, and then be quiescent for a corresponding length of time. Two adjacent glomeruli could be situated within a few microns of each other, and yet the blood flow in one could stoy completely, with no perceptible alteration in the blood flow of the other. This phenomenon of intermittence is a peripheral affair, as it persisted after destruction of the brain and spinal cord. Krogh found that when blood ceased to flow through a given capillary, it disappeared from view. The Pennsylvania workers found that when the blood flow stopped abruptly in a single active glomerulus, the corpuscles would stagnate for an instant, and then disappear. It is possible that glomerular capillaries are under a similar regulatory influence to capillaries in general. If so, a relationship may at last be established between certain forms of hypertension and alterations in kidney structure.

It follows from the above discussion that all the glomeruli do not function at once. The number of glomeruli acting at any given moment may determine the extent of kidney damage inflicted by circulating toxins or organisms.

They found that substances which depress the circulation, such as pressor doses of adrenalin or pituitrin lessen the number of glomeruli that are active. Substances which stimulate the circulation, such as hypertonic saline, caffeine, increase the number of glomeruli that are active with consequent diuresis. At least one variety of diuresis is, therefore, due to increase in the number of acting glomeruli. Small doses of adrenalin decrease the flow of blood through the rabbit kidney, increase its volume, and increase the volume of urine. This triad is explicable by assuming that adrenalin acts on the efferent glomerular vessel, and this constitutes another variety of diuresis.

By introducing a capillary pipette into the capsule of Bowman, Richards and Wearn collected a sample of glomerular filtrate. It contained urea, sodium chloride, and glucose in approximately the same concentration as in the blood. Bladder urine collected at the same time contained no glucose, and under certain circumstances, no sodium chloride. The *modern* theory thus receives striking confirmation. The work of Bieter and Hirschfelder, who employed the same technique, confirms the view that urine is excreted in dilute form through the glomeruli, and concentrated by the tubules.

Assuming that these results obtained in the frog hold good in the human, it follows that the human kidney is not one single structure, but a composite organ, and like the eye of a fly, consists of many individual units. A human kidney contains approximately two million glomeruli, and the daily twelve hundred cubic centimetres of urine are probably formed by four million little kidneys. This conception is quaint as well as epoch making, and promises much in our interpretation of renal pathology.

The student is referred to the following references:

Richards, A. N., and Plant, O.H.: Am. J. Phys., 1922, lix, 144, 184, 191.
Richards, A. N., and Schmedth, C. F.: Am. J. Phys., 1922, lix, 489.
Wearn, J.T.: Ibid, 490.
Richards: Kidney Function, Am. J. Med. Sc., 163:1, 1922.
Bieter and Kirschfelder: Am. J. Phys., 68:326, 1924.

TWO COMMON FRACTURES

G. E. Richards, M.B.

Chief of Department of Radiology, Toronto General Hospital

In the course of the routine daily work in a large Radiological and Physio-therapeutic Department, one has the opportunity to observe some points of interest regarding certain simple, or at least very common, fractures and injuries about joints. A few of these appear to be fundamental, and yet are not infrequently overlooked. No apology

should be necessary, therefore, if emphasis is placed upon certain very homely facts. The present paper will be limited to Pott's fracture and Colles' fracture.

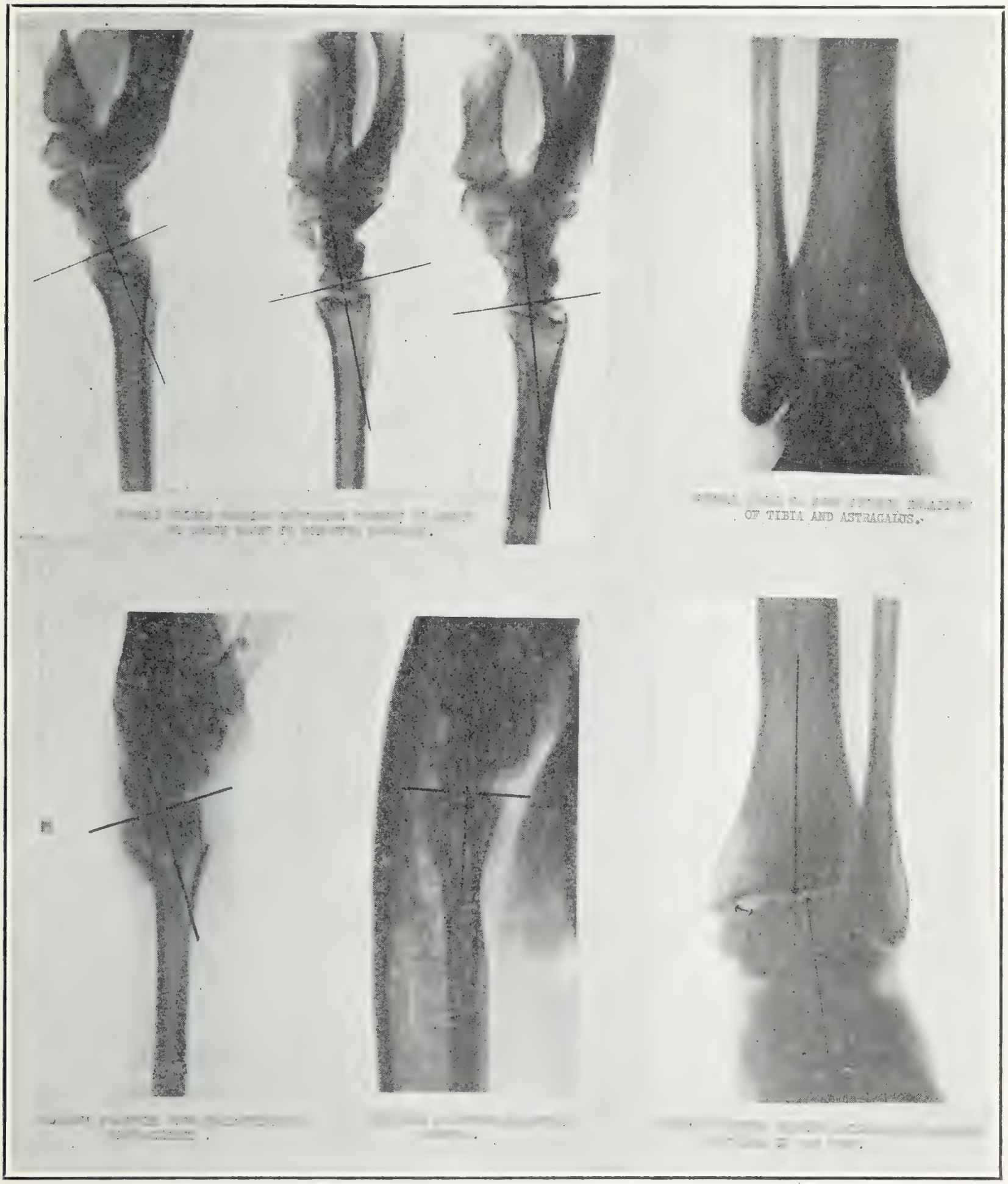
Pott's Fracture

Without entering into an academic discussion as to what Potts originally described, it may be stated briefly that the common injury about the ankle which is included under this term, consists of three factors:

1. A fracture of the external malleolus.
2. A fracture of the internal malleolus.
3. A semi-dislocation outwards of the foot in relation to the lower articular surface of the tibia.

It matters little how much attention is paid to the correction of the two former if the latter is left undiscovered or, if discovered, is not adequately and completely corrected. Since it is one of the commonest of fractures, it is somewhat surprising to observe the frequency with which this phase of the condition is overlooked. We have used the term "semi-dislocation", and yet this wording does not accurately describe the displacement which occurs. This may vary from an exceedingly slight lateral slip which does not measure more than an eighth of an inch, to a degree of displacement in which the centre of the astragalus is well over to, or even beyond, the external edge of the articular surface of the lower end of the tibia. We are scarcely concerned here with the latter, since a displacement so obvious is not likely to be overlooked. But in those in which the slip has been slight only careful examination of the X-ray plates will reveal the true state of affairs. For this purpose the most useful X-ray examination consists in single antero-posterior and lateral plates, the greatest care being taken to ensure that the tube is centred exactly over the ankle-joint, and that there is no rotation of the limb as the exposure is made. Under normal conditions in such a plate, a line drawn vertically downwards through the shaft of the tibia and through its lower articular surface will pass also through the centre of the upper articular surface of the astragalus.

If there is lateral displacement present this fact may be recognized by a study of this simple point and one other, namely, the spacing which exists between the inner malleolus and the adjoining portion of the astragalus. Normally these surfaces are separated only by the cartilaginous surfaces covering them, and this appears on the plate as a thin space not more on the average than 1/16th inch. If this space is definitely wider than this it may be considered evidence of some lateral slipping. In any doubtful case it may be well to examine the opposite joint. If the slipping amounts to ¼ inch, as very often is the case, and this displacement remains unreduced, the result will almost invariably be a stiff, painful ankle, with some degree of limitation of movement. Furthermore, if the displacement is not recognized until several weeks have elapsed, the space fills in with organized exudate or fibrous tissue to such an extent that complete correction later will be exceedingly difficult, and may be impossible.



OF TIBIA AND ASTRAGALUS.

For this reason it is most desirable that an X-ray examination should be made immediately after the reduction, even if this has been done under fluoroscopic control. In the vast majority of cases the two *fractures* which are present are correctly dealt with, but in a certain number the third factor will be overlooked unless it is deliberately sought for and corrected.

Colles' Fracture

In this case also there are certain facts which may be regarded as fundamental. For our present purpose the chief interest centres in a study of the lateral projection of the lower end of the radius as it is seen normally, and in a Colles' fracture. A series of wrists were examined and a few of these are shown in the illustration. In the majority it will be seen at once that the lower end of the radius bearing the articular facet is directed slightly forwards, and if a line is drawn joining the two points of this articular surface, and then from the centre of this a perpendicular is erected, it will not pass up the centre of the fore-arm as is commonly supposed, but will emerge through the back of the wrist. The exact angle thus formed varies slightly with individuals, but generally is found to be from three to ten degrees *forward*.

In a typical Colles' fracture the small lower fragment is displaced backward, and the long upper portion of the shaft is impacted into it in greater or less degree. This in turn is the cause of more or less comminution on the dorsal aspect.

The necessity of disimpacting these fractures has been taught for many years, and is only referred to here in order that the urgency of this procedure may be again emphasized. If the impaction be allowed to remain, the result will almost always be a stiff painful wrist with limitation of dorsi-flexion, just as in the ankle, failure to correct the lateral displacement results in a persistently painful joint.

The ideal reduction, of course, would include complete anatomical restoration of the parts, and in this case requires that the lower fragment be replaced in such a manner that the articular surface is again directed forwards three to ten degrees. That this has, in fact, been accomplished, can only be determined by subsequent X-ray examination (even if the reduction here also has been done under fluoroscopic control). We repeat this adominition because of the frequency with which we are asked to omit this re-examination and trust to the fluoroscope. The latter is exceedingly useful, but is not sufficiently accurate in matters of such fine judgment, and even if this were true the fluoroscope leaves no permanent record. It is important for all concerned that there be a permanent record of the fracture as it was at the completion of the reduction as well as before.

SIR JAMES MACKENZIE

A. H. Walker, '29

The death of Sir James Mackenzie, which occurred several weeks ago, removed a man whose accomplishments in the fields of Medicine and cardiology placed him in the forefront of the ranks of the world's greatest men. It is doubtful if any one man has contributed more to cardiology than Sir James Mackenzie. Certainly none have succeeded him in ability to interpret the symptoms of cardiac disorder, in terms of structure and function. The greatness of this man is best realized, when one recalls that, just previous to his death, the Mickle Fellowship, given the man who was considered to have made a most striking contribution to science, was awarded him by the University of Toronto.

To the medical student who has not yet studied physiology, knowledge of Mackenzie's work will awaken a greater interest in the subject, to the more advanced student and the graduate, who almost daily are using his findings, the story of his eventual triumph over the difficulties under which he worked, cannot help but be encouraging.

Sir James Mackenzie graduated in Medicine from the University of Edinburgh in 1878, at the age of twenty-five. After spending a term of one year as house physician at the Royal Infirmary at Edinburgh, and after taking a short course at Vienna, he began to practise at Burnley. For twenty-eight years he remained in this Lancashire town. It was during his residence there and while engaged in all the activities of a general practitioner, and with a hospital appointment, that he carried out investigations which led to the introduction of the polygraph as a means of studying pulse irregularities. From the very first of his medical career he was impressed with the inability to interpret the pulse variations, and by his life-long efforts was able to change the chaos of the nineteenth century into the order of the twentieth. It was long before his researches yielded anything in the nature of a general message to science, and those who have followed his career know that the time which he spent in collecting notes and verifying conclusions, would have terrified a man less nobly absorbed by his double determination to arrive at truth and to do good.

His first published paper was on "The Study of Sensory Symptoms Associated with Visceral Disease". This article was followed by "The Venous and Liver Pulses and the Arrhythmic Contraction of Cardiac Cavities". From the beginning Mackenzie felt that if the generally accepted foundations of cardiac therapeutics were to be labelled as insufficient or unsound, unimpeachable evidence must support the challenge. Hence it was not till 1902 that his book "The Study of the Pulse", appeared. In 1907 he settled in London and quickly took his place as one of the foremost consultants, in the diseases of the heart. He was appointed physician to the Mount Vernon hospital and some years later became head of the cardiac department of London Hospital. His greatest book, "Diseases of the Heart", was published in 1908. It was widely read and accepted and can now be obtained in German, French and Italian.

One of the greatest ambitions of his life was to encourage research into the beginnings of disease, and with this object in view he resigned his London appointments in 1918 and established at St. Andrews an Institute for Clinical Research.

During the years intervening between his removal to St. Andrews and his death, he published many notable articles and books on the nature, cause, and treatment of diseases of the heart, as well as on other medical topics. By this time the magnitude of the work which he had done in connection with diseases of the heart was recognized all over the world. He had thrown light upon the functional derangements of the heart in an amazing manner; and further, gave indications for logical diagnosis, prognosis, and treatment. The arrhythmias and the manner in which the heart responds to varied conditions that may be brought about by infectious diseases or by the exhibition of drugs, were made the subjects of special research. He devised instruments convenient for the purpose of modifying the sphymograph so that simultaneous with a radial tracing the record of other movements connected with the circulation or respiration could be noted.

Sir James Mackenzie was a man of striking personality. A friend gives this estimate: "He was hard-headed and warm-hearted, quick in temper and in sentiment, genial, hospitable and possessed of an invigorating blunt humour. Schooled by long experience in the sensibilities, reservations, obstinacies and frailties of patients, he was an astute and shrewd physician, who rarely failed to inspire an unlimited and merited confidence."

When carried away with his own views, he was apt to leave the impression that lines of investigation other than those adopted by him, were useless. In this way his diatribes against stethoscopes, electro cardiographs, valvular murmurs, and morbid anatomy were apt to be misleading to those who did not understand him.

He believed that medicine was taught in the wrong order and that the simpler and common diseases should be demonstrated by the out-patient department, while a junior physician would be quite competent to teach in the wards on the fully developed diseases. He saw, as few of his day saw, where clinical knowledge ends and ignorance begins, and never hesitated to define the limits of knowledge. It is said that although an authority in clinical medicine, he would frequently use the words "I don't know".

Many great honours were conferred upon Sir James Mackenzie. Among them was his election as Fellow of the Royal Society, in 1915, "for his researches into the heart in health and in disease". In the same year he was knighted and later was appointed physician to the King, in Scotland.

It is unnecessary to eulogize this famous doctor, or to dwell upon the recognition which his work received. His accomplishments stand alone and form an excellent illustration of what a desire for complete knowledge, coupled with perseverance and genius, can effect.

Sir James Mackenzie is indeed one of few men who have "raised a monument more enduring than bronze". The magnitude and practical value of what he has done, will perpetuate his name and cause it to be honoured, so long as the science of Medicine exists.

THE SIGNS OF ANAESTHESIA

W. Easson Brown, M.B.

Research Fellow in Therapeutics, University of Toronto

One of the chief difficulties of the young anaesthetist is the difficulty of accurately determining at any desired time the exact depth of anaesthesia at which his patient may be. This, however, is not particularly difficult, providing the anaesthetist is thoroughly familiar with the various signs of anaesthesia. The accompanying chart has been worked out with the purpose of showing the various signs of anaesthesia during the different stages and phases in such a way that these different signs may be compared with one another with ease.

Anaesthesia is divided into four stages. The first stage is from the commencement of the anaesthetic until consciousness is lost; the second stage, from the loss of consciousness to the loss of corneal reflex; the third stage, from the loss of corneal reflex until poisonous signs commence; and the fourth stage, that of toxic manifestations. The third stage for the purpose of description is divided into four phases which are purely arbitrary and roughly divide the stage into four equal parts.

During the first stage the patient is conscious. Therefore providing the anaesthetic is given in a manner not to cause discomfort or unpleasantness to the patient all signs should be normal as is shown. The induction of an anaesthetic should never be hurried. If the patient complains of a choking sensation the anaesthetic is being administered too fast.

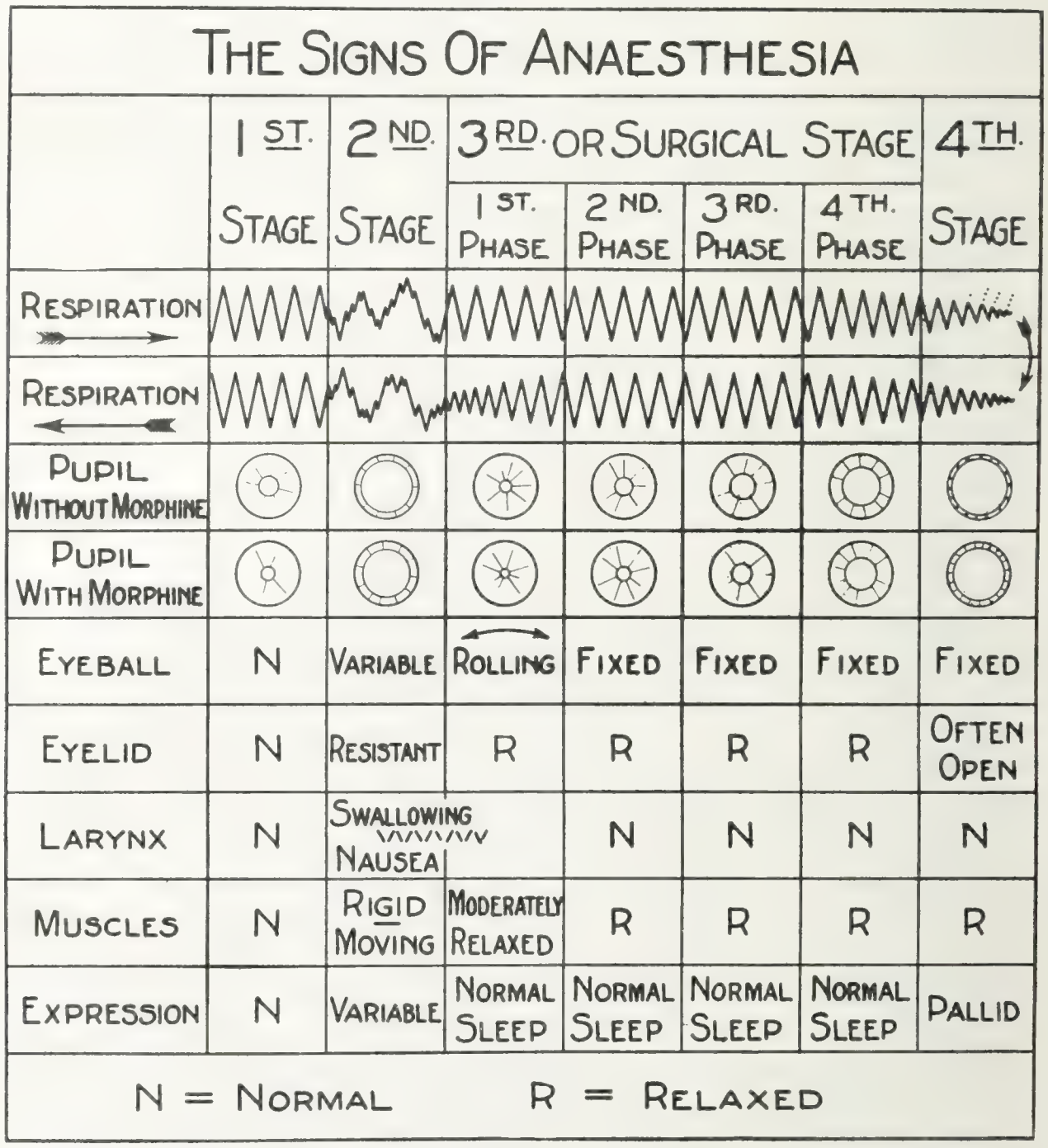
As the patient loses consciousness and enters the second stage there may be great variations in the respiration. Breath-holding is quite common, although sometimes fast regular or irregular respiration is seen. The pupil is dilated and often reacts somewhat to light. The eyeball actions may be variable. Movement is common or positions of external or internal rotation may occur. Any endeavour to open the eyelid is usually met with resistance. There is practically always swallowing, and frequently nausea. The muscles show signs of a removal of cerebral inhibition in struggling movements or exaggerated rigidity. The expression is variable and the face may be greatly contorted.

As the anaesthetic is continued the patient gradually reaches the third or surgical stage of anaesthesia. The breathing again becomes regular, usually somewhat deeper and faster than during consciousness. The pupil is quite small and often oscillating slowly from side to side. All muscles are moderately relaxed. A continuation of the anaesthetic in sufficient concentration causes only slight changes as may be seen in the second phase. The respiration is unchanged. The pupil varies little in size but is much more apt to be stationary. The muscles are relaxed, providing there is no cyanosis. The expression is that of normal sleep. As the depth of narcosis is increased, very little change takes place in the respiration but there is a noticeable increase in the size of the pupil. The other signs show little change. As the fourth phase of the third stage is reached, which is the deepest degree of anaesthesia required, the

breathing is often somewhat shallower. The pupil is moderately dilated and other signs are unchanged.

As the toxic stage is reached the breathing becomes very noticeably shallow and there is frequently a tendency toward prolonged expiration. The pupil is widely dilated. The eyelid sometimes falls open and the expression is bewildered and the face pallid.

Referring to the chart it will be seen that there is a second horizontal line for respiration. This line refers to respiration as the anaesthesia is becoming lighter. This line follows the upper line closely until the first



phase of the third stage is reached, when it is seen to taper off, the respirations becoming quite light. If respiration alone was the guide the anaesthetist might be in difficulty as to whether the patient was in this light stage of anaesthesia or in the toxic condition of the fourth stage. The matter is easily settled by reference to the pupil. In the light third stage it is small and often active; in the fourth stage it is fixed and widely dilated. Attention is also drawn to the size of pupil in the second stage and in the fourth stage. Both are seen to be widely dilated. This is often a cause of worry. However, if the other signs, such as breath-

holding, straining, struggling, swallowing and nausea are taken into account, there is very little possibility of mistaking them for absolute relaxation and a pallid countenance. The second line of pupil diagrams is inserted to show that patients who have received a normal dose of morphia, followed by a normal reaction, have a gradual dilatation of the pupil as anaesthesia is deepened, but that this dilatation occurs with slightly deeper degrees of anaesthesia.

The phases, as has been noted, are for purposes of description and chiefiy in regard to depth required for various surgical procedures. For example: In the first phase of the third stage, which is a splendid degree of anaesthesia to maintain, operations requiring only slight relaxation should be attempted by the beginner. In maintaining this phase the little finger must be kept on the thyroid cartilage so that the slightest movement may be noted and a sufficient amount of anaesthetic given to keep the patient out of the second stage. It must be noted here that in order to arrive at this stage of anaesthesia it is, of course, unnecessary to put the patient into a toxic stage and then allow recovery up to the first phase. It is sufficient if the anesthesia is deepened to about the third phase before the amount of anaesthetic is lessened and the patient allowed to become lighter. Operations on extremities, mastoids, etc., can be done in this phase of anaesthesia.

In the second phase lower abdominal and such operations as require moderate relaxation may be done.

Gall bladder procedures require the third phase, while work involving the rectum usually needs a fourth phase anaesthesia.

In conclusion one may say that this is only a very rough résumé of the principal signs of anaesthesia. Nevertheless, they are signs that everyone who endeavours to administer an anaesthetic should know. Knowing these, the young anaesthetist may approach his patient, if not with entire confidence, at least with the knowledge that he is not setting his patient adrift like a rudderless ship on a sea of unconsciousness, but that he will be able to guide him past the dangerous reefs, and bring him once more to the harbor of conscious safety.

THE CARE OF PREMATURE INFANTS

JEAN R. DUNCAN, '25

From a paper read before the Alpha Omega Alpha Faternity, Feb. 2nd, 1925.

A premature may be defined as a child started more than three weeks too soon upon its struggle for independent existence. The spark of vitality in them appeals to the physician just as the smoldering fire does to the firemaker. The flickering flame of life is fanned and fed with great care during the first days and weeks, though many cynics may declare it is only to postpone their deaths for a short time. Ylppö has proven that to save the premature for one year is to give it equal chances with the full term child. So one may find interest in the ways by which these "unfinished tasks of nature" have been nurtured to the enjoyment of a full and healthy life.

The first duty is to combat the tendency to hypothermia, for the high proportion of surface to volume in the very small must represent a large area of radiation. To this end labour should be conducted in a warm room, 75° Fahrenheit, the child should be received in warmed clothes, and the washing fluids should be of blood heat. In connection with this group, Hess brings up the perennial question of leaving the tying of the cord until all pulsations cease, claiming that thereby the frail bit of humanity is given the benefit of an additional thirty to sixty cubic centimeters of blood.

Respiration must now be facilitated. This is first encouraged by using a mucus tube to clear the air way, then artificial respiration is exerted rhythmically for one minute by pressing on the thorax with the thumb and palm of the right hand twenty times per minute. The warm bath at 105°F. is tried for five minutes, and sometimes the stimulation of cold water sprinklings is tried. Finally oxygen, given through a catheter 200 bubbles in the minute may be effective, or again mouth to mouth insufflation, with adequate precautions taken against infection. While the heart beats the attendant should be encouraged to continue resuscitation measures.

Clothes wisely chosen to fulfil the following principles may greatly reduce the heat loss by conduction and radiation.

1. They should fit snugly so that there is no thick air blanket about the child.

2. The material should be a poor conductor of heat, such as French flannel.

3. The garments should be unconfining.

4. The design should be such as to enable dressing to be done with the minimum of handling.

These requirements are fulfilled by a knitted wool shirt and canton flannel diapers, worn over the band, which will be worn longer by a premature than a full term child, because the cord takes from 8 to 10 days to mummify. Over this a double French flannel Red-Riding-Hood cape is worn, which fits closely and covers every part but the face. Absorbent pads may replace the diaper in the very small, to reduce the handling, a point which must be greatly stressed in the management of prematures.

External heat has long been considered 'the most effective' weapon against the heat loss in these undeveloped babies. As early as the seventeenth century we read of one Fortunio Siceri[1], whose father, being a physician, was not discouraged at finding himself with a son come too early, and no larger than a man's hand; but with heat and carefully selected food, he saved for the world a life of eighty years, crowned with scientific achievement. Since the ingenuity of the French has been aroused to save prematures and thereby offset their falling birth rate, many designs of incubators have been used, most interesting of which are Deuncé's double bath and Rommel's type.[2] Incubation was very like asphyxiation when closed designs were used, such as those of Tamier and Finkelstein. A very efficient and practical model is that described by Dr. Brown.[7] This is a twenty-four inch laundry basket lined by silence cloth, with non-absorbent cotton over the bottom to the depth of eight

inches, over which oil cloth is put, being sewn in through the wicker sides. The bedding is arranged over this. The external heat is supplied by six well stoppered bottles, which are changed frequently enough to maintain a temperature of eighty to ninety degrees, the filling water being at one hundred and ten. To control this heat a thermometer is folded in the bedding and read frequently. This individual bed is kept in a room at 70°-75°, and a relative humidity of about fifty-five per cent.

It is the super heated room, in a hospital, which is the most efficacious means of getting excess heat for these debilitated infants. The source of heat has to be a constant one, and can be arranged by getting live steam from the sterilizers. The ventilation comes in from one side of the room, through double windows, so constructed as to let the air in at the bottom of the outer and into the room through the top of the inner. Dishes of water are placed on the radiators to supply the desired moisture. At the Burnside, almost complete isolation is obtained by the use of glass partitions placed between the cribs, reaching up about four feet. The cribs themselves are of ordinary design with cot covers to exclude all draughts. The temperature of the room is kept at about 85 degrees, the thermometer being so placed as to be read without entering the room. All these precautions are taken so that the premature's temperature may be stabilized between 97 and 97.6 degrees, which means a variation in the room temperature of not more than 6 degrees.

The dangers of "refrigeration" are encountered before the premature is brought to hospital and during their transference thereto, and hence the importance of institutions having "Transportation Incubators", such as are described by Hess.[1] This necessity is impressed by the figures for the Sick Children's Hospital for the year 1924, when it was found that of the 76 admitted, 54 died, but 24 of this number did not live beyond 36 hours, due largely to the chilling received during transit.

The feeding of the premature is of primary importance, and perhaps this ought to be regarded as meeting the gastro-intestinal complications found in these infants, owing to the incompleteness of their physiological development. The great problem is to get sufficient ingesta absorbed through the inadequate organs to meet the demands. This is illustrated by the stomach capacities as given by Hess[1]; at twenty-six weeks in utero the capacity is 2 drachms; at forty weeks, nine and a half. Thus is presented the mechanical difficulty of having enough food absorbed (1) to sustain life; (2) to maintain weight; (3) to allow for growth. The type of food to be used is unquestionably breast milk. All available statistics point to the natural food as the only solution. Most convincing among these figures as those of Dr. Godfrey Pisek, for New York in 1916, in which 85 per cent. of all the infant deaths were among the artificially fed. Or to put it more concretely, four artificially fed children die to one breast fed. Eden shows that while 31 per cent. of breast fed prematures die, 50 per cent of those on artificial mixtures succumb. Various authorities have suggested alternatives, such as Albumin Milk, advocated by Grulee,[3] Chymogen Milk, Lactic Acid Milk, Butter Soup, and skimmed and buttermilk preparations. Rotch[4] of California went so far as to say all food for prematures should be prepared in the laboratory so as to fit the specific needs. The benefit of the natural

milk does not need to be urged further, but the source is of perennial interest. The early arrival of the infant means that were it to be fed by its own mother, it would receive colostrum over quite a long period, and this is productive of diarrhoea and is particularly bad for the premature. A wet nurse is one answer to the problem but reputed to be a very uncomfortable one for the family. An alternative is the establishing of Breast Milk Bureaus such as are in existence in Detroit and Toronto. Either in the selection of a wet nurse or of the source of the human milk supply great care should be exercised to prove a clear bill of health. The ideal milk for the premature comes from a mother with a month old baby. Manual expression is carried out with scrupulous care from the standpoint of asepsis and the milk is refrigerated and handled with the greatest possible precautions.

The methods of feeding and the interval must now be decided. These are both judged by the degree of development shown by the child. In the very weak, cyanotic infants, Grulee recommends the use of a catheter, not only to overcome the difficulty of the inefficient swallowing apparatus, but also to conserve the strength of the premature. A fruit spoon, or a medicine dropper, or a Breck feeder may be used where the suctorial powers have not yet appeared. It is well to put the child to the breast as soon as possible for thereby the maternal flow is sustained at the required level, and it is simpler and safer than manual expression. Most authorities agree on the three hour interval in feeding, except Grulee, who uses the gavage method every four hours. The infant is not fed during the first twelve hours, and then a half and half breast milk and sterile water mixture is given. Fluids are used between feedings, sterile water and lactose, 1 to 5 per cent. being usually employed.

The initial weight loss is a significant factor in estimating the prognosis and as it has been shown that it is proportionately much higher in the premature than in the full term child, it places a definite emphasis on the need to push fluids and food in this group. The smaller the infant the higher the percentage of the birth weight lost. A method of conserving the body fluids is by substituting an oil rub for the daily bath and thereby insulating the body surface to some extent. The danger as to food seems to be definitely one of underfeeding rather than overfeeding, and so the caloric demands per pound run high, the average of figures given by eleven authorities being 72 calories a pound—the range being from 44 to 88 calories and the highest amount fed aggregating 133 calories in the day. The infant is maintained at the intake of about 50 calories, but must have between 65 and 100 to gain. In applying these rules care must be taken not to overstep the tolerance of the delicate organism. Also it must be remembered that the initial loss in the premature persists for 12 to 14 days and the birth weight is not regained until the twenty-first day.

The care described above is the means which must be taken to give every healthy premature born at six months the chance to live. In addition the following complications must be kept in mind, as those to which the child "born early" is particularly predisposed. Dr. Simpson's survey of the prematures, which had passed through Burnside during the

last ten years, gives digestive disturbances as the greatest cause of death, with respiratory conditions such as bronchopneumonia and atelectasis taking second place. Besides, such conditions as anaemia, rickets, tetany and mental defect were found to some degree. It is not within the scope of this paper to suggest ways of meeting all these complications, and methods of treatment are just touched upon.

Cyanotic attacks constitute the "bete noir" of those attending the premature and it is to be recalled how they are caused. (1) By atelectasis, which is combatted by instituting a daily cry, effected by the least possible amount of trauma. (2) By digestive disturbances, which should be prevented by sane feeding. (3) By lack of development of the respiratory centre. Whatever the cause, the use of warm mustard baths is indicated, one ounce to a gallon of water. Perhaps the use of oxygen more completely meets the demand of the condition when the third cause is considered, at least such is the claim of Bakmin[6], who suggests that the gas be administered through nasal catheters, or better, by using an oxygen chamber. This last he regards as a prophylactic measure against these attacks if the child is exposed for half hour periods.

There are other complications, which do not swell the mortality figures so much as they increase the number of disabled and undesirable citizens. It is arresting to find that 95 per cent. of the prematures under one year of age, examined by Dr. Simpson, showed signs of rickets, while 20 per cent. of them had had cod liver oil. In the second year 80 per cent. showed the condition and 40 per cent. had had the oil. It is difficult to trace all the factors producing this result, but it remains that the shortened time in utero greatly reduces the calcium content in prematures, for 85 per cent. is stored within the last three or four months of foetal life. Besides, there is an additional calcium loss in these infants owing to the higher proportion of faecal output. This is particularly true with a high fat diet. Perhaps this explains the earlier appearance of rickets and spasmophilia in prematures than in the full term infant, usually about two months sooner. So it is advised that cod liver oil be started by the fourth week, with a daily dose of eight minims gradually worked up to thirty. Frequently the flaring of the epiphyses is not present and this gives support to Bengt Hamilton's[10] claim that the condition is one of osteoporosis, which he has shown will respond to calcium chloride administration. Tetany is sometimes distinctive in this group in that it does not show the same electrical hypersensitivity. A third deficiency disease, scurvy, appears early likewise, and orange juice is advocated as soon as the second month.

Anaemia is almost invariably present, which persists throughout the early years, the Burnside Survey having shown it to be a handicap up to the fifth year. This anaemia must be of the chlorotic type, for Eden has shown a polycythaemia to be present in the prematures. The condition is best met by giving a transfusion during the first year. Hess suggests iron carbonate in ¼-½ gram doses three times a day, started at the fourth week. Iron may be obtained through a diet containing egg yolk and vegetable soups with spinach.

Ylppö thinks that prematures are particularly prone to mental defects

and it is found that the percentage of mongols is higher than in children born at full time. However, other forms of deficiency are due to birth injuries, and these are more prevalent the smaller the infant. It is hard to state that prematurity, per se, is a cause of various degrees of idiocy when Newton, Hugo and Renan were among those born early.

In conclusion we must realize that age, weight, initial loss, length, tissue turgor, and congenital disease all affect the prognosis, but more than these is the care the physicians are willing to give. In Toronto in 1914 there were 18.4 premature deaths per 1,000 living births, as compared to 19.5 in 1923. Last year 257 prematures died in this city; such a rate of death among our Medical Faculty would wipe us out in three years. It is a case of our lives for the prematures. Ballantyne has said that he can quote mortality figures from authorities on Prematurity, ranging from 10% to 90% and yet during the last ten years at Burnside 303 prematures have been cared for and 280 have been discharged—a 7% mortality.[9]

REFERENCES

[1]Julius Hess, M.D.: "Premature and Congenitally Diseased Infants."
[2]Hess, in Abt. Vol. II: "Care of Prematures."
[3]Gruleé: North American Clinic. 1923; p. 477.
[4]Rotch, article by Cook: Arch. of Paediatics. xxxviii; 201. (1921).
[5]Gruleé: Surgery, Gynaecology and Obstetrics. xx; 234. (1915).
[6]Bakmin: American Journal Diseases of Children. 1923/25/157.
[7]Brown: Arch. of Paediatrics. (1917) p. 609.
[8]Cruickshanks: Public Health Journal, January, 1925.
[9]Dr. Simpson's Survey of Burnside Cases of Prematurity—read before Toronto Academy of Medicine.
[10]Hamilton: Amer. Journal Diseases Children. xx; 316. (1920).

MULTIPLE PREGNANCY

E. Clark Noble, M.A., '25

(From a paper read before the Alpha Omega Alpha Fraternity on Feb. 2, 1925.)

The subject of multiple pregnancy should be of interest to the general practitioner from two standpoints. In the first place, the etiology, diagnosis, and management of labour is of interest. And again, the subsequent care of such infants is of great importance, for it is well known that in from 60-70% of all multiple births the infants are premature. In this short treatise, twin births are taken as the most common example of multiple pregnancy.

Etiology

The frequency with which multiple pregnnacies occur would seem to vary with the fertility of the populace at large, statistics showing them to be relatively more common in Russia and Ireland, and low in France.

Multiparae, and especially those with large families to their credit, are more liable to multiple pregnancies, which probability, according to De Lee, increases after the age of thirty-three. Without doubt, there is an hereditary tendency, which is nearly always transmitted through the female, and rarely if ever through the male, although the intermarriage of twins is said to increase the liability to multiple births. It is a relatively common phenomenon for a woman to give birth to twins on more than one occasion. Geisler quotes the case of a woman physician, who in 33 years of married life gave birth to 13 twins and 6 triplets—44 children in all—her two sisters having 41 and 26 children respectively.

Incidence

Twin pregnancies are fairly common, most authorities agreeing on their occurrence in about 1 in 90 cases. The incidence of triplets is about 1 in 8,000, and quadruplets 1 in 400,000 cases.

Types

Two varieties are encountered; one, the most common, results from the fertilization of two distinct ova, which are derived from one or two Graafian follicles, and is known as the binovular type. The other, the uniovular type, results from the fertilization of a single ovum, which may contain two blastoderms, or one which subdivides into two.

(a) *Binovular Twins*—The two ova are usually fertilized at or about the same time; occasionally, however, this may have occurred at different periods, and the two foetuses be of different ages. Thus we may have (1) Superfecundation, a condition where fertilization has taken place during the same intermenstrual period, or (2) Superfoetation, where fertilization has occurred during two different intermenstrual phases. Here the age differences may be considerable, but it is definitely limited, for after the fusion of the decidua vera and capsularis, around the third month of pregnancy, fertilization cannot occur. Clinically these two conditions are difficult to differentiate, as twin foetuses of identical ages may be subject to such variations in nutrition that one is of apparently greater age than the other. In binovular twins, which are from five to seven times more common than uniovular, there are two distinct chorions and amnions and no matter how closely the two placentae are approximated, no communication exists between them. The sexes may or may not be the same, and Ballantyne asserts that there is no foundation in the popular belief that the female born co-twin with the male is sterile. The children are usually fairly robust, and not so subject to malformations as the uniovular variety.

(b) *Uniovular Twins*—Here the arrangement of placenta and membranes varies, but generally one finds two chorions, two amnions, with but one large placenta, or more rarely two placentae between which there is more or less communication. During the later months of intra uterine life, the two amniotic cavities may coalesce and the two foetuses develop in one cavity. The children are usually underweight and not very robust, always of the same sex and bearing a marked resemblance to each other.

Malformations, even progressing to monstrosities are more frequent in this type. While the latter subject is, I think, outside the scope of this paper, one of the commonest varieties might be mentioned; these are what are known as "double monsters", and are examples of uniovular twins in which the common blastoderm has not divided into two. Here we encounter the thoracopagus, dicephalus, syncephalus and so forth.

Course of Twin Pregnancies

In between 60 and 70 per cent. of cases, premature labour ensues. In a series of 3,000 cases, Miller found 60% were premature and underweight, and even if carried to full term, many were considerably underdeveloped. Thus in at least three-quarters of all cases, the ultimate care of twins resolves itself into the care of the premature. The complications of pregnancy, such as the toxaemias, are found to be relatively more frequent and severe.

Diagnosis

Granted the diagnosis of pregnancy has already been established, the further diagnosis of multiple pregnancy is by no means so easy as might at first thought be considered. Until the fifth month it is almost impossible, and from then until the term it becomes relatively less difficult, but I think the fact remains that twin pregnancies are diagnosed most frequently only after the birth of the first child.

On palpation suggestive points to be sought for are overdistention of the uterus and multiplicity of limbs, while more diagnostic findings are two distinct breeches or heads. Mistakes may readily arise from a large fibroid present in the uterine wall, a fair sized ovarian cyst or the presence of hydramnios. This latter condition occurs fairly frequently in twin pregnancies, but its onset is characteristically fairly abrupt about the 28th week of gestation.

Auscultation affords exact evidence if one can be certain, of two hearts beating at different rates. Offermann considers two distinct funic souffles to be diagnostic.

Vaginal examination is of little assistance until labour has commenced. On the whole the diagnosis of plural pregnancy is of slight consequence, provided that one always is equipped for the delivery and reception of two infants. Sometimes, however, in order to maintain one's pride as a diagnostician, one might wish to advise the family of their impending catastrophe.

Management of Labour

Labour, as already pointed out, is usually premature, on account of the overdistension of the uterus. Bannister considers 36 weeks to be an average gestation period for twins.

The relative frequency of various presentations is given by Munroe Kerr as follows:

Double Cephalic	38%
Head and Breech	21%
Breech and Head	14%
Double Breech	10%
Transverse Lie (Second Fetus)	13%

With the onset of labour it is not uncommon to find that the membranes have ruptured prematurely, due to the lack of coaptation between the presenting part and the pelvic brim. An examination should be made to discover the possibility of prolapse of the cord. A careful examination to verify the prenatal findings of the presentation of the fetus should be made. In a primipara, according to Bannister, it is unusual to find any presentation but a vertex or breech, at least in the first fetus.

The first stage of labour is usually somewhat prolonged, by reason of a degree of uterine inertia brought about by overdistension. The cervix usually dilates rapidly, and as the fetuses are small, no delay is experienced during the second stage. Delay may occur from two vertexes attempting to engage at once. Under such conditions, one should, using full anesthesia, push the presenting part of one fetus up out of the pelvis and attempt by pressure to engage the presenting part of the other.

After the birth of the first child the precaution should be taken of ligating securely the placental end of the cord, in view of the possibility of an anastamosis between the two placental circulations, especially in twins of the uniovular variety. An abdominal and vaginal examination should be made to determine the lie and presentation of the remaining fetus, and to rupture the remaining membranes. The second child is usually born after fifteen minutes rest, and is followed shortly by the placenta. Occasionally in binovular twins, the first fetus is followed by its own placenta, and as there is no anastamotic circulation, the other fetus is in no immediate danger. Profuse hemorrhage or distress on the part of the remaining twin is an indication for its immediate delivery.

Post partum hemorrhage is more liable to occur by reason of the larger placental site, and a certain degree of uterine inertia. The occurrence of "locked twins" as a complication of labour is so rare that it may be disregarded.

BOUND COPIES OF THE JOURNAL

Subscribers who wish to do so may have their copies of *The Journal* bound by The University of Toronto Press. The cost of such binding is reasonable, provided the copies which it is desired to have bound are left with The Press.

Back copies of *The Journal* may be obtained from the Secretary's office, Medical building, with the exception of copies of Vol. I, No. 1, December, 1923.

FACULTY NOTES

Omitted It is regretted that this section was omitted from *The Journal* in the last issue. This occurred owing to the fact that the copy was not available at the time we went to press.

Rumour Denied Professor Wasteneys' friends are glad to know that the rumour that he is leave the University of Toronto is without foundation.

Graduating Picture The group picture of the Graduating Year was arranged this year by Aylette and is generally considered very satisfactory. Each member of the graduating year received a copy printed from a cut of the picture, and paid for from class funds.

Alumni Association All members of the graduating year are urged to take advantage of the offer of the Alumni Association whereby they receive their first year's membership in the Alumni Association, including a year's subscription to the *University Monthly,* for one dollar, instead of the usual three dollar fee.

Subscribe Now Members of the graduating year will be given an opportunity to subscribe to *The Journal* before leaving the University and their support is requested. The value of *The Journal* will be as great after as before graduation.

Dean Primrose Honoured The congratulations of all are due to Dean Primrose for having recently been made a Fellow of the Royal College of Surgeons, without examination. This is a very rare honour and one in which the University of Toronto may therefore take pride.

Editor For Next Year Readers of *The Journal* will be interested to know that Mr. N. A. McCormick, of the Class of 2T6, has been appointed editor-in-chief of *The Journal* for next year. The outgoing board of editors wish for him every success in his efforts.

Torontonensis The new, enlarged, improved edition of Torontonensis is now issued. It is a volume of which every graduate may feel proud and which every one should appreciate fully, after having paid for it on the instalment plan over the past six years. The new shape and art work in this year's Torontonensis show considerable improvement over the Torontonensis of other years, however excellent they have been, and it is a volume which will show up well on the waiting room table, among all the out-of-date numbers of *The University Monthly, Goblin,* and *Vanity Fair.* The book in its new form is the result of the efforts of the Editor, Mr. W. A. Higgins, of Forestry, and practically all credit must be given to him. Mr. Higgins was at one time managing Editor of *The Varsity.*

INDEX TO VOLUMES I AND II.

(NOTE:—The articles are indexed under Volume, Number and Page; *e.g.*, Volume 1, number 4, page 20, is written I; 4; 20. In the second volume the pages have been numbered consecutively through the whole volume.)

A

Abdomen, Time Factor in Emergency Conditions of the;—
H. E. Clutterbuck ..... I; 4; 3.
After Graduation What?—*Ed.* ..... II; 4; 113
Anaesthesia, Rectal, in Labour—*Ed.* ..... I; 3; 3.
Anaesthesia, The Signs of—*W. E. Brown* ..... II; 7; 257.
Anatomical Science, The Development of—*K. G. Gray* ..... I; 2; 33.
Angina Pectoris, The Nature of—*Ed.* ..... II; 1; 4.
Appendicitis, Historical Outline of—*G. C. Stephens* ..... II; 4; 132.
Article, On Writing an—*Ed.* ..... II; 2; 39.
Articles, Dr. Rudolf's—*Ed.* ..... II; 2; 39.
Atoms and Ions—*E. F. Burton* ..... I; 3; 28.

B

Breast, Tumours of the—*A. Primrose* ..... I; 5; 8.
Bricks without Straw—*Ed.* ..... II; 4; 114.
Bright's Disease, The treatment of—*H. S. Hutchinson* ..... I; 1; 7.
Build Broad before you Build High—*Ed.* ..... II; 6; 187.

C

Carcinoma of the Stomach—*H. W. Wookey* ..... II; 5; 152.
Cardiological Practice, On Cardinal Principles in—*Thomas Lewis* ..... I; 2; 24.
Cerebral Hemispheres, Origin of the—*Ed.* ..... I; 4; 4.
Charlatanism—*Ed.* ..... II; 7; 229.
Chemistry in Medicine—*W. R. Lang* ..... I; 2; 24.
Chemistry in Medicine—*W. H. Martin* ..... II; 6; 222.
Chemistry in Medicine, The Era of—*Ed.* ..... II; 5; 152.
Cholecystography—*Extract* ..... II; 6; 221.
Clarke, Dr. C. K., In Memoriam ..... I; 3; 6.
Clubbed Fingers—*Ed.* ..... I; 4; 4.
Constipation, Purgatives in Chronic—*R. D. Rudolf* ..... II; 4; 128.
Course, An Unscheduled—*E. S. Ryerson* ..... II; 2; 61.
Course We Have Had, The —*Ed.* ..... II; 6; 186.

D

Dementia Praecox, Some Notes on—*C. K. Clarke* ..... I; 1; 10.
Dermatitis Venata, A New—*Ed.* ..... I; 4; 4.
Development, Some Problems of—*W. H. Piersol* ..... I; 5; 21.

Diabetes Mellitus, Dietetic Treatment of—*W. R. Campbell* .....I; 4; 24.
Diathermy—*Jean Masten* .....II; 5; 173.
Digitalis in Chronic Heart Failure—*R. D. Rudolf* .....II; 1; 21.
Doctor's Diary, From the—*Extract* .....II; 7; 236.
Do You Ever Think?—*Ed.* .....II; 4; 115.
Dyspepsias, Classification of—*Ed.* .....I; 1; 27.
Dysphagia, The Causes of—*E. C. Noble* .....II; 6; 209.

E

Eclamptic Toxaemia, Treatment of—*H. B. VanWyck* .....I; 2; 11.
Electricity in Medical Education, The Study of—*L. Gilchrist* .....I; 1; 24.
Electrocardiogram, The—*J. Hepburn* .....II; 7; 233.
Encephalitis Lethargica, Contagiousness of —*Extract* .....II; 3; 96.
Encephalitis Lethargica, Some of its Problems—*Ed.* .....II; 3; 73.
Encephalitis Lethargica, Symptoms of—*G. W. Howland* .....II; 3; 100.
Endocrinology, A Report of a Lecture on .....II; 2; 63.
Eventually, Why Not Now?—*Ed.* .....II; 5; 149.
Evolution of Man, Present Day—*J. W. McArthur* .....I; 3; 13.
Examinations, How to Pass—*Ed.* .....II; 7; 230.
Expectorants—*R. D. Rudolf* .....II; 5; 170.
Eye Conditions in Pregnancy—*C. E. Hill* .....II; 5; 176.

F

Fractures of Long Bones, Compound, Treatment of—*C. L. Starr* ..... I; 3; 7.
Fractures, The Healing of—*R. M. Janes* .....II; 3; 106.
Fractures, Two Common—*G. E. Richards* .....II; 7; 251.
Future of *The Journal,* The—*Ed.* .....II; 7; 231.

G

Gallie, Dr. W. E., Hunterian Professor—*Ed.* .....I; 3; 3.
Garth and the Apothecaries—*A Graduate* .....I; 1; 23.
Goitre—*C. B. Horton* .....II; 2; 48.

H

Harvey, William—*A Graduate* .....II; 3; 97.
Head-Colds, The Cause of—*Ed.* .....I; 5; 6.
Heart Disease, Exercise in—*G. E. Smith* .....I; 5; 18.
Hemorrhagic Disease of the New Born—*D. V. Clark and A. D. Kelly*..II; 5; 188.
Heredity and Eugenics—*J. H. D. Smith* .....II; 5; 166.
Hippocrates, Case Notes of —*Extract* .....I; 3; 35.
Hunter, John Irwin, M.D.—*A. H. Walker* .....II; 4; 126.
Hunter, John,—The Naturalist—*A Graduate* .....I; 4; 22.
Hydropic Degeneration of the Islets of Langerhans—*D. J. Bowie*.....II; 5; 155.
Hypnotics, The Use of—*R. D. Rudolf* .....II; 2; 54.

I

Infant Feeding, Butter Soup in—*A. M. Courtney* .....I; 1; 17.
Inhabitants of Man and their Migrations, Some—*A. H. Walker*.....I; 4; 36.
Insulin, Discovery and Preparation of—*F. G. Banting and C. H. Best*....I; 3; 24.
Insulin, The Physiological Action of—*J. J. R. Macleod* .....I; 2; 6.
Interneships for Women—*Extract* .....II; 7; 232.
Intestinal Intoxication, Acute, In Infants—*A. Brown and G. Boyd*.....I; 1; 6.
Introduction, By Way of—*Ed.* .....II; 1; 3.

J

Jaundice: Normal and Pathologic Physiology——*R. P. Mackay* .....II; 5; 158.
Jaundice: Some Clinical Considerations—*J. E. Walker* .....II; 6; 199.
Jenner, Edward—*A Graduate* .....I; 5; 28.

L

Laboratory, Pathological, Technique of—*W. L. Robinson* .....I; 5; 38.
Leeuwenhoek—Pioneer Microscopist—*M. E. Hall and R. K. Haight*....II; 2; 59.
Literature for Medical Students—*Ed.* .....I; 5; 3.
Liver Function Tests (I)—*I. R. Vaile* .....I; 3; 30.
Liver Function Tests (II)—*I. R. Vaile* .....I; 4; 33.
Living Matter in Biology and Medicine—*W. H. T. Baillie* .....I; 5; 42.

M

Mackenzie, Sir James—*A. H. Walker* .....II; 7; 255.
Man's Long Quest—*Review* .....I; 1; 25.
Mark Twain Said—*Extract* .....II; 6; 225.
Medical Discussion Groups—*Ed.* .....II; 3; 75.
Medical Education—*Ed.* .....II; 2; 37.
Medicine, The Art of—*Extract* .....I; 2; 5.
Medicine in the Old Times—*Extract* .....I; 5; 6.
Medicine, The Successful Practice of—*A. G. McPhedran* .....I; 5; 14.
Meltzer-Lyon Test in Gall Tract Diseases, The—*G. L. Fair* .....I; 2; 27.
Menstruation—*F. W. Marlow* .....II; 6; 193.
Menstruation, The Physiology of—*D. M. Low* .....II; 6; 197.
Mental Disorders, Focal Infection in—*Lillian Sher* .....II; 2; 57.
Mental Testing—*E. A. Bott* .....II; 6; 204.
Monstrosities, Causation of—*J. C. Watt* .....II; 3; 90.
Muscle Tone, Anatomical Basis of—*E. A. Linell* .....I; 3; 21.

N

Nephritic Test Meal, The—*V. J. Harding* .....I; 1; 20.
Nerve, Anatomy of Vth Cranial—*E. A. Linell* .....II; 4; 137.
Nerve Centres, Development of—*Ed.* .....I; 2; 3.

Nerve Impulse, Nature of—*J. M. D. Olmsted* ..... I; 1; 14.
Nemon Theory Substantiated, The—*Ed.* ..... I; 5; 4.
Nobel Prize—*Ed.* ..... I; 1; 4.
Note Taking, Some Points on—*R. P. Mackay* ..... II; 1; 26.

O

Onlooker, The (poem)—*Amy Lowell* ..... II; 6; 203.
Osler, Sir Wm., A Maker of Medicine ..... I; 5; 13.
Our Patrons—The People—*T. W. G. McKay* ..... II; 4; 117.
Ovarian Hormone—*Ed.* ..... I; 1; 4.

P

Para-smallpox—*Extract* ..... II; 5; 175.
Pathological Individuals and Their States—*F. E. Perry* ..... II; 3; 103.
Photosynthetic Comedy A—*F. E. Slosson* ..... II; 1; 30.
Pneumothorax—*Ed.* ..... I; 3; 4.
Posterior Position, The—*K. C. McCarthy* ..... II; 4; 141.
Pregnancy, Multiple—*E. C. Noble* ..... II; 7; 264.
Pregnancy and Pulmonary Tuberculosis—*Extract* ..... II; 1; 12.
Premature Infants, The Care of —*J. R. Duncan* ..... II; 7; 259.
Preventive Medicine, State Control of—*T. H. Belt* ..... II; 1; 13.
Psychiatrist in Court, The—*Ed.* ..... II; 1; 5.

R

Recipes for the Diabetic ..... I; 5; 31.
Reflexes, Visceral—*R. P. Mackay* ..... I; 4; 18.
Render Unto Caesar The Things Which Are Caesar's—*Ed.* ..... II; 5; 151.
Reticulo-Endothelial System—*Ed.* ..... II; 4; 146.
Rickets, Role of Calcium in—*Ed.* ..... II; 2; 39.

S

Scarlet Fever—Etiology of—*Ed.* ..... I; 5; 5.
Scarlet Fever, Modern Conception of—*A. H. Graham* ..... II; 2; 64.
Science and Ethics—*Ed.* ..... I; 4; 3.
Skin Diseases, Diagnosis of—*E. J. Trow* ..... II; 5; 179.
Sleep—*D. L. McLean* ..... II; 4; 122.
Something Canadian Medical Students Need—*Ed.* ..... II; 1; 3.
Spine, Fractures of—*J. H. Couch* ..... II; 1; 17.
Spleen, Recent Knowledge of—*Extract* ..... II; 7; 247.
Sprains—*H. W. Wookey* ..... II; 7; 245.
Surgical Dressings—*A. B. LeMesurier* ..... I; 3; 18.

T

Talk to Senior Medical Students—*A. Fisher* ..... II; 5; 163.
Tenosynovitis, Acute Suppurative—*W. S. Muir* ..... II; 6; 216.
Treves, Sir Frederick—*Ed.* ..... I; 2; 4.
Tuberculosis, Diagnosis of Pulmonary—*T. G. Heaton* ..... II; 7; 237.
Tuberculosis, Etiological Studies—*Extract* ..... II; 1; 31.
Tuberculosis, Gold Treatment—*Ed.* ..... II; 6; 185.
Tuberculosis, Intestinal—*A. E. Broome* ..... II; 2; 41.
Tuberculous Adenitis, Diagnosis of—*Extract* ..... II; 1; 28.
Typhoid Fever, Prophylaxis and Treatment—*J. C. Goodwin* ..... II; 3; 77.

U

Ulcers, Progress of Peptic—*H. A. Bruce* ..... II; 1; 5.
Ultra Violet Therapy—*K. McMurrich* ..... II; 6; 224.
Ultra Violet Therapy and Tuberculosis—*Ed.* ..... II; 3; 74.
Urinary Secretion, Physiology of—*J. Markowitz* ..... II; 7; 249.
Urinary Tract, Diagnosis of Diseases of—*R. J. McComb* ..... II; 6; 212.

V

Vaccines, Some Principles in Employment of—*H. B. Maitland* ..... I; 4; 13.
Vaccines, Some Principles in Employment of—*H. B. Maitland* ..... I; 5; 47.

SILVER IODIDE IN A NEW ROLE **NEO-SILVOL** COLLOIDAL SILVER IODIDE

NEO-SILVOL contains 20% of Silver Iodide in colloidal form. It makes an opalescent, milky solution in water, one that leaves a scarcely perceptible stain on drying. These solutions do not irritate the skin or mucous membrane to which they are applied, and their germicidal activity has been fully demonstrated by bacteriological and clinical tests.

The indications for the use of Neo-Silvol include conjunctivitis, gonorrheal ophthalmia, naso-pharyngeal infections with or without sinus or antral complications, cystitis, and acute and chronic urethritis of gonorrheal origin.

One medical author reports one injection (by catheter) of 5 cc of a 10% solution of Neo-Silvol is giving him excellent results in the acute cystitis of young children. He also finds it one of the best remedies he has ever used in the treatment of pyelitis, introduced by way of the ureter.

Neo-Silvol is equal to carbolic acid as a germicide, and twenty times as fatal to the gonococcus.

NEO-SILVOL is supplied in 1-oz. bottles of the granules and in 6-grain capsules, 50 to the bottle. One capsule makes one fluid drachm of a 10% solution.

Write for a sample and literature.

PARKE, DAVIS & COMPANY

NEO-SILVOL IS INCLUDED IN THE N. N. R. BY THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION

CLASS OF 2T5

Before Graduation Subscribe to

THE UNIVERSITY OF TORONTO

MEDICAL JOURNAL

for

NEXT YEAR

Price $1.25